GAS POISONING IN MINING AND OTHER INDUSTRIES

GLAISTER AND LOGAN

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BY

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WITH PLANS, COLOURED PLATES, AND THIRTY-SIX OTHER ILLUSTRATIONS

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PREFACE.

In the following pages an attempt has been made to focus within reasonable limits our knowledge of the subject of poisoning by carbon monoxide and other gases arising chiefly in mining and other industrial pursuits. The subject has been the theme of valuable papers, some monographs, and a few theses by many authors abroad, and while the task of acquiring a knowledge of these has not been an easy one, it has proved of considerable interest and profit. On the clinical side, there has not been the same attention shown by home authors. If we except the writings of Haldane, Mott, and one or two others, there is not much more to show.

While this volume cannot pretend to have overtaken exhaustively the views of all these authors, an honest endeavour has been made, to present the principal work which has been done.

The writers of this volume have had considerable experience, each from his own standpoint, in the matters discussed therein, and during the acquirement of that experience they felt, as doubtless have others, that a volume dealing comprehensively with industrial gas-poisoning would meet a want which has been experienced by not a few, especially with reference to accidents in mining arising from foul air and noxious gases, and to other industries in which, for various purposes, gas is now extensively employed.

In view of the large number of cases which annually come before Courts of Law for compensation for accident under the Workmen's Compensation Act and other Acts of Parliament, the writers believe that such a book as this is calculated to prove helpful to practitioners of law as well as to practitioners of medicine.

In the preparation of the volume the authors have to acknowledge most gratefully assistance which they have received. To some they are under obligation for suggestions with regard to portions of the text, to others for assistance in reading the proofs, and to others for the use of blocks to illustrate the text. To all they now tender their sincere thanks.

In particular, they desire to thank Professor Burns, Professor of Mining in the Royal Technical College, and Professor T. K. Monro, Professor of Medicine in the University of Glasgow, for valuable suggestions; Dr Walker of Edinburgh for most helpful service in reading

the proofs and preparing the volume for the press; Dr F. W. Mott, F.R.S., London, for the use of blocks illustrative of the intimate pathological changes in the central nervous system; the publishers for their ready willingness to meet the wishes of the authors; and the following firms for the use of blocks illustrative of rescue apparatus, etc., viz.:—Messrs Jacobson; the Sauerstoff-Fabrik, Berlin; Siebe, Gorman & Co.; the Power-Gas Corporation, Ltd.; Wallach Bros. Ltd.; and others.

The bibliography in the appendix, together with the numerous references in the text, may prove a source of assistance to the future enquirer in his quest of the literature of the subjects treated in the volume.

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CHAPTER I.

INTRODUCTION.

ALTHOUGH in Ironworks, Steel works, and Collieries, hundreds of cases of poisoning by gases must occur almost weekly, with every now and then one which ends fatally, there is surprisingly little literature on the subject, since very few medical men connected with Ironworks and Collieries put their experiences on record. In this work we shall base our remarks on cases which have come under our own notice in Ironworks and in numerous Collieries, and in connection with compensation work, at the same time availing ourselves of the experiences of medical friends who have treated a considerable number of such cases.

During the last few years the use of gas in industrial occupations has enormously increased, and with the greater perfection of the gas-engine, its uses have become greatly extended. It has been well said that while the nineteenth century has been the era of the steam engine, the twentieth will be that of the gas engine. In a large iron and steel work, for example, the gases from the furnaces and producers are used for many different purposes, so that what was formerly looked upon and termed waste gas is now regarded as a most valuable product. In many works, indeed, gas is now used in the different stages to convert the ore into the finished steel products ready for the market. The gas may be conveyed long distances, and, as it is used for so many purposes, we can quite readily understand that there are ample opportunities for accidents to occur. Gas poisoning cases then, both major and minor, are greatly on the increase, and will probably still further increase. The *Lancet*[1], commenting on this, said, "Carbon monoxide is rapidly becoming a modern terror, for undoubtedly the number of victims to CO poisoning is decidedly on the increase."

This subject is a most important one in these days of compensation for accidents, as both acute and chronic poisoning by CO have certain remote effects which can only be properly understood with more extended knowledge and much fuller records of cases than we at present possess. At the present day, Judges and Sheriffs, adjudicating upon such cases, have very little to guide them in their dècisions, as the evidence given before them by doctors and experts is not infrequently so contradictory and so clouded as to be almost valueless for guidance. Not only so, but the experts cited to give evidence

[1] *Lancet*, Vol. I., 1903, p. 258.

may not themselves have had any personal experience of such cases. Some of them, as a matter of fact, seem to have but a very vague idea of what the remote effects of CO poisoning really are; or if, indeed, such effects exist at all.

In the Report of the Departmental Committee on Compensation for Industrial Diseases four cases of gradual poisoning by furnace gas, brought forward by Dr Judson Bury and Sir Thomas Oliver, were discussed. In these cases, in which unconsciousness at no time supervened, the chief symptom was multiple neuritis, and although there appeared to be little doubt that some constituent of the furnace gas was the direct cause of the illness, the Committee concluded thus—"The subject, however, is by no means free from obscurity. Had a gas, which is so common as CO, possessed noxious qualities of this character, we should have expected that the fact would have been recognised long since and in many more than four cases. The evidence that has been so far collected we cannot consider sufficient to establish that poisoning, either by carbonic oxide or by other gases evolved in furnaces, can be regarded as a trade disease." In reply to this we aver that such cases are by no means so uncommon as the evidence heard by the Committee would lead us to believe. We believe that the principal reason why more is not heard of the unusual effects of CO poisoning is that many medical men have not, so far, been educated to recognise them; thus illnesses of a mysterious nature and origin, which are probably caused by gas poisoning, are frequently put down to totally different causes.

Several most important questions present themselves for special consideration, as, for example, that of pneumonia and other pulmonary diseases following exposure to CO, for compensation cases are frequently occurring in Court where pneumonia is declared to have followed, and to have been caused by, the inhaling of the fumes resulting from the use of explosives in mines, or by the inhaling of coal gas, owing to defective gas connections in a house, or from like causes.

Again, the question of sudden death resulting from poisoning by CO is not sufficiently understood. We remember the case of a young man where the evidence pointed to death having been caused by the inhalation of the gaseous products of combustion from heavy blasting. In this case the Sheriff and medical assessor held that death was due to apoplexy, and thus to natural causes. In connection with this, it is interesting to note that fifty or sixty years ago, a common verdict found in cases of gas poisoning, owing to the fact that the post-mortem examination disclosed cerebral hæmorrhage, was "Death by Apoplexy, accelerated by breathing impure air."

Two other most important points are, whether prolonged working in bad air may produce heart disease in miners, and whether the

possibility of work in such an atmosphere may lead to serious consequences, and even to death, in men who have certain cardiac lesions.

The physiological and experimental side of carbon monoxide poisoning has been very ably and fully worked out by Dr Haldane, this part of the subject being, in a manner, complete; but we cannot say the same about the clinical side of the question. Sufficient attention has by no means been paid, at least in English literature, to the remote effects of CO poisoning. From the perusal of most of the literature on the subject, one is apt to run away with the impression that when recovery from CO poisoning does take place, such recovery is always complete. That, as we shall see, is far from the real state of affairs; it often takes months for the effects of the poisonous action of CO gas on nerve tissue, the heart, etc., to wear off, and in certain cases the damage done is permanent. Much more attention, moreover, is now being paid to the Diseases of Industries and Dangerous Trades, regarding which there is already a very considerable body of literature, and consequently we are entitled to hope for more thorough and careful attention being paid in future to such cases of gas poisoning as we are about to discuss.

Brouardel[1] in pointing out how important to the medical jurist is a knowledge of the action of CO on the brain, quotes the following interesting case, which occurred not so many years ago in Paris:—

A woman of uncertain reputation was discovered by her husband in open adultery, and he threatened to leave her. A few days afterwards, people passing the house observed the woman making violent gestures at her window, shouting that her husband was dying, and asking the people to inform her brother, who worked in a neighbouring factory. On entering the house they found her husband and brother quite dead. The husband was lying in bed stiff, and the dead body of the brother was found on the threshold of the door leading to the cellar. The woman was unable to give any explanation of what had occurred; she appeared to be drunk. This condition lasted during the next day, when she appeared in Court. The warder under whose charge she was, remarking on her curious condition, said "You must have been terribly drunk on Sunday for you have not recovered three days after." This persistent state of intoxication struck the Magistrate who examined her, for he added the following note at the end of his examination. "We note that in the course of examination the woman D. is in a marked stuporous condition. Her replies are obtained only with difficulty, and on asking questions we found that occasionally she answers, at other times she does not, while occasionally she replies to something which was not asked." In the course of the trial, one witness said, "About three weeks ago I went to the house of D. at mid-day. I found the husband,

[1] Brouardel: *Les Asphyxies par les Gaz, etc.*, 1896, p. 261.

who was alone downstairs, heating milk for his wife who was lying ill in her room above. Suddenly he was seized with shivering, sat down on a chair, and lost consciousness. I then called his wife, who came. In a short time she also settled herself in a chair, leaned on the table, muttering 'My poor man, how you are suffering,' then she seemed to fall asleep, but she did not lose consciousness. She remained absorbed in thought, and did not in any way attempt to help her husband, which seemed to me strange. The husband soon regained consciousness and commenced to vomit, but in a short time was able to take a little coffee. In the afternoon the brother went out of the room and vomited several times at the foot of the stair. Having asked him what was wrong with him, he replied that he did not know, that certainly he and D. were worse than his sister, allowing me to infer that the latter was drunk." The same witness saw the husband again. He came down dressed, as if he had just risen, was stupid-looking, and told witness that he was ill, "just as in the last attack." He trembled, staggered when he walked, and was very pale. There were no signs in the house showing that excessive drinking had been going on, and no proper evidence of such was produced in Court. The singular attitude of the patient, her embarrassed answers, and her absence of energy in defending herself, had such a strong bearing against her that she was found guilty and condemned to penal servitude for life. In this case everything pointed to death by CO: the skin had a rosy tint and the blood was a brilliant red. The experts (one a Professor of Chemistry in the Faculty of Science, and the other Professor of Clinical Surgery in one of the first schools in Paris) ignoring these and other facts pointing to death by CO poisoning, declared that the two men had been poisoned. The post-mortem examination showed inflammation of the bowels with submucous hæmorrhages, and therefore they thought that death had been caused by a non-corrosive substance which had caused death by absorption. The experts did not find any trace of a mineral or alkaloidal poison. They considered it possible that poisoning was due to powdered cantharides, because a very small fragment of what seemed to be part of the integument of an insect was found in the vomited matter. Later, several accidents occurred in the same house; and after seven years the case was revived. It was then found that the two men had died from poisoning by CO which had come from the fumes of a lime-kiln which adjoined the house. Undoubtedly the woman herself was a victim to CO, which had destroyed her brother and husband, and the ignorance of the experts of the peculiar action of CO on the nervous system placed her in this terrible position, and made her the victim of a serious miscarriage of justice.

The following case of *Lesser* [1] shows the peculiar torpor and mental

[1] Lesser: "Les Empoisonnements," *Atlas de Médicine Légale*, 1890, p. 137.

state produced by CO, and how this may be wrongly construed. On the 20th January 1881, a man and his wife and daughter went to bed about seven o'clock in a room containing a stove. At five o'clock the husband woke up, and although he was in a dazed state, he was able to light a lamp. He found his daughter quite dead, and his wife in a comatose state; but instead of procuring help he lay down on the bed beside his wife. At eight o'clock he awoke again, and suddenly the idea struck him that their serious plight was caused by charcoal fumes from the stove, and so he attempted to open the valve of the stove but failed. Then he reseated himself on the edge of the bed for a few hours, roused himself sufficiently to go out, visited a restaurant, had something to drink, and then went back to his home without speaking to anyone. Next day, the 22nd, he went out again, and on returning declared that he did not leave his bedside till the morning of the 25th. On being questioned, he said he thought that his wife died on the 24th; so that he had waited eighteen hours after her death before reporting it. He was arrested on the 25th, and was then so excited and troublesome that the police thought he was drunk, although after careful examination it was found that this was not the case.

The following is another case of poisoning by the fumes of burning coke where two, of three persons exposed, died. The survivor was apprehended on suspicion of having caused the death of the other two. A woman, Honorah Hill, staggered into a neighbour's house about five p.m., and told the neighbour that both her father and mother were dead. On entering the house, the old people were found lying in bed quite cold. It was noticed that there was no odour of smoke or gas in the room. The daughter staggered about so much, and appeared so excited, that the police thought she was drunk, and as her statement of what had occurred was so unsatisfactory as to raise further suspicion, she was arrested. The statement she made was that on the night before, refuse coke was used for making a fire, and that it burned very badly and filled the house with smoke. The mother suffered from bronchitis, and the smoke so aggravated her cough that the accused threw up the window and soon afterwards went to bed, leaving her father and mother chatting by the fireside. When she woke up in the morning, it was to find her father dead and her mother dying. She could not, however, move her lower limbs, and she screamed for assistance several times but no one heard. It was late in the afternoon before she was able to make her way downstairs. Her father was sixty-one years of age, and three weeks previously he had fallen down into the cellar and broken his ribs. The wife was fifty-one and had suffered from bronchitis. They frequently quarrelled. Dr Bury conducted the post-mortem examination. On the man he found several bruises of recent origin; the lungs showed old mischief,

both being bound to the chest wall by old adhesions, and there was also old-standing disease of the lungs; the 9th, 10th, and 11th ribs were fractured near their angles, and a swollen bruise was found on the back over the seat of fracture. Dr Bury held that the fractures must have taken place a few days before death. He would not give a positive opinion as to the cause of death. There was no direct evidence that death had been caused either by violence or by asphyxia, and he suggested that the contents of the stomach should be examined. He was also unable to state the cause of death in the woman. Dr Cullingworth afterwards examined the bodies and concluded that there was no evidence of death from violence. The fractured ribs must have been caused two or three weeks before, in view of the amount of repair present. He held that death was due to CO, and that the condition of the lungs of the parents made them succumb more easily. The evidence of CO was found in the blood by Dr Waters of Owen's College. The result of the gastric analysis was negative. The woman was discharged.

A proper knowledge of the action of CO is necessary for the treatment of poisoning cases—as ignorance of its action in lowering the body heat is not unlikely responsible for a certain number of cases ending fatally through improper treatment.

We cannot proceed further without calling attention to the debt of gratitude which underground workers owe to Dr Haldane for his efforts to place on a sound scientific foundation the analysis of the air of mines, and the action of the various gases on men breathing them. Notwithstanding the fact that Dr Haldane has on every possible occasion taken the opportunity of placing his views, experiments, and knowledge before the various mining societies, and has written several popular mining papers solely for the benefit of the mining community, many managers and mine officials are still ignorant of this subject, while it is impossible to give the necessary information to the miners on account of their well-known dislike to attend mining and other classes and lectures instituted for their benefit. To bear out what has been stated, let us quote some remarks of Lord Kingsburgh, the Lord Justice-Clerk, with regard to CO poisoning in a pit in a case which came before the Court of Session (an appeal from the Sheriff Court) in November 1908. In giving judgment the Lord Justice-Clerk said that he found the case to be "not unattended with difficulties. It must be kept in view that the danger from carbon monoxide was one which till lately had not made itself prominent in pit management. Science had recently brought it into the region of known things, but so little was known in regard to it that, as one of the witnesses said, if mine owners were looking for managers who had full knowledge in regard to carbon monoxide, they would find a very minute

percentage who had any information or experience in regard to it." These remarks in some measure might with justice be extended to medical men, for as students they have to depend for their knowledge of CO poisoning upon works of medical jurisprudence, in which the information is generally of the most meagre description and sometimes incorrect. It is a great pity that Dr Haldane's work is not known to medical men as it ought to be. Perhaps this is to a certain extent due to the fact of his work appearing in Blue Books, mining manuals such as *Investigations of Mine Air*, in the Transactions of various Mining Institutes and Societies, and in Journals such as the *Journal of Physiology*, which are certainly not read by the average general practitioner. We should also add that very few Continental contributors to this subject have appreciated the value of Dr Haldane's work.

Dr Haldane has also put on a proper scientific basis rescue work after explosions in mines, and he has made it much less hazardous by showing how far one can travel in a tainted atmosphere, and by calling attention to the value of small animals as a danger signal, the use of which he was the first—in our country at all events—to advise.

But before approaching the subject from the standpoint just indicated, we shall give a brief account of the formation of these poisonous gases, as without such knowledge it would be impossible to discuss this question satisfactorily. At the same time we shall confine our attention as much as possible to those points which have a practical bearing on the subject.

CHAPTER II

General Description of Methods of Working Collieries.

Coal is reached by means of a shaft or shafts which are sunk to the coal strata, another shaft being generally placed near it in order to allow of a system of ventilation to be completed as soon as possible. These shafts are connected by roads being driven in the coal. The two principal methods of working the coal are "board and pillar," "pillar and stall," or the "stoop and room" system as it is called in Scotland, and the "longwall"; any other modes of working coal being simply modifications of these. The method of working depends upon local conditions, more particularly upon the thickness of the coal strata. Other conditions will decide which is to be used as, for example, "troubled" field, presence of water, inclination of strata, character of roof, etc. In the "longwall" method the coal is removed in one working only, the roadways being carried through the goaf or space from which all the coal has been taken. The "stoop and room" process, with all its modifications, is used when the "longwall" is unsuitable. First of all, places are driven in the solid coal so as to divide the area of coal into large rectangular pillars or blocks of coal of the size laid down in the plans. The narrow places driven are called "stalls" or "rooms" in Scotland—in splint coal, for example, the dimensions of these measure nine to twelve feet. The second working consists in the removal of the pillars. When these are removed, this part of the mine where all coal has been taken out is called the "goaf." The extraction of the "pillars," as one can readily understand, is therefore a most important and dangerous part of the work.

The amount of coal which a miner can detach and fill into hutches depends greatly upon the kind of seam at which he is working, as well as other conditions, but four or five tons per day may be taken as an average output. There are long lines of working faces where the men are distributed at anything from five to forty yards apart, the distance depending on the method of working. The hutches are pushed along by the "drawers" to the main roads, where the ponies or horses (depending on the height of the roads) pull them to places where the haulage is completed, perhaps by engine power. Many roads form self-acting inclines. A mine may be then divided into the following sections viz:—shafts, working-faces, and the goaf or worked-out places. The main roads are made of suitable height, and are securely timbered or supported by

"trees" to prevent "falls" which cause such an appalling number of mine accidents; and in places where the roof is particularly bad it may be arched with bricks.

Ventilation of Mines.

It is necessary to say something regarding the ventilation of mines, as it has an important bearing on our subject In this we shall in no way trespass upon ground which belongs essentially to mining experts, but only deal with a few points which bear directly on the health of those working below, points which, therefore, appeal specially to medical men.

Great progress has been made recently regarding our knowledge of the different causes which operate to interfere with proper ventilation, and very great strides have been made in the mechanical methods of supplying a proper quantity of air to mines. Not only so, but our knowledge regarding the pollution of the air in mines, the sources of this pollution, as well as the character of the gases and their action, have all been made the subject of expert inquiry, and the knowledge gained subjected to scientific criticism. Notwithstanding this work, however, a great deal still requires to be done to eliminate the many factors of danger which exist underground. That this is necessary is shown by the following statement made in the Report of the Royal Commission on Accidents in Mines (1909); "When we began to consider what further provision could be made, it became apparent to us that the existing information with regard to dangerous or harmful impurities present in the air of mines was very scanty" (page 72).

The Pollution of Air in Mines.

The air in mines is polluted by different gases, such as black-damp, methane, etc., which are given off from the strata; by CO_2 from men and horses, and from the gases generated in the burning of lamps; gases which are formed as the result of oxidation of the coal, timber, organic matter; by the formation of CO_2, CO, etc., in varying quantities from explosives; and by Sulphuretted Hydrogen which is given off by the action of water, etc., on iron pyrites, and which in underground fires gives rise to the smell in "gob-stink."

In all these cases, it is not only the presence of adventitious gases in the air which constitutes the danger, but also the fact that the vital constituent of the air, oxygen, is at the same time being used up.

Other substances in the air of mines which add to the danger are the fine particles of coal-dust, and also of chemical substances which result from firing gunpowder, etc.

From this short account of the gases, etc., which pollute the air in a mine, one recognises how essential it is to prevent their accumulation, to dilute them so as to render them harmless, and to replace the amount of oxygen which has been used up. This is what is meant by the ventilation of mines.

The *Coal Mines Act*[1] says that "An adequate amount of ventilation should be constantly produced in every mine, to dilute and render harmless noxious gases to such an extent that the working places of the shafts, levels, stables, and workings of the mine, and the travelling roads to and from those working places, shall be in a fit state for working and passing therein."

This definition of what constitutes adequate ventilation is much too vague, for one can read divergent meanings in the word "adequate." Our knowledge now-a-days, regarding the properties of poisonous gases and the quantities sufficient to produce harmful results, is much more extended and complete. Besides, our knowledge of the results of the different explosives is much more exact, so that what was formerly looked upon as adequate ventilation is not now regarded as such. Conditions naturally vary very much in different mines. Take, for example, the presence of fire-damp. We now know that even small percentages of fire-damp may cause explosions if certain other conditions are present. The only practical way at present of estimating the amount of fire-damp is by means of the lamp. Many hold that the ventilation of a mine should be considered inadequate when the presence of gas is detected by an ordinary safety lamp in the main return ways. Putting this into percentages of fire-damp, from our knowledge regarding what can generally be detected by the ordinary fireman, we may say that, if in fiery mines not more than 2 per cent. of gas is found in any part of the returns in a ventilating system, this may be regarded as adequate ventilation. If at the working face there is more than 2½ per cent., the place should be fenced off. According to the 1909 *Report of the Royal Commission on Mines*, the following should be the standard of ventilation. "Every reasonable endeavour should be made to maintain such a standard of ventilation as to prevent the appearance in any open and readily accessible position of a fully formed cap on the lowered flame of the safety-lamp, and men must not be allowed to work or pass where this standard is exceeded. Enquiries are being made as to the percentage of fire-damp which constitutes a fully formed cap."[2]

No hard and fast rule can be drawn regarding the volume of air to be supplied, as here again we are face to face with two or three great difficulties, for it is not an easy matter to measure accurately the amount of air which passes. There must be many conditions in operation in a

[1] General Rule 1 of the Coal Mines Regulation Act, 1887 (section 49).

[2] Royal Commission Report, 1909, p. 206

pit to cause marked fluctuations in the amount of air current; and another most important practical point is, that it is often difficult to come to a correct knowledge of the sectional area of the roadways and workings.

In order to conform to the Regulation regarding adequate ventilation quoted above, it is generally held that each man requires 100 cub. ft. of air per minute in non-gaseous mines, and from 250 to 400 cub. ft. in fiery mines. Some mines have very much more than the latter figure—but generally speaking, the volume ranges from 100 to 600 cub. ft. The volume of poisonous gases given off from the strata varies, as already mentioned, in different mines, and even in the same mine it depends largely upon the amount of exposed surface of exudation. Roughly speaking, the quantity of air supplied should bear the same ratio relative to the number of men and horses as to the exposed surfaces of exudation, or, to be more scientifically correct, to the area of workings and the coal output. Another point to be emphasised is this; that greater stress must now be laid upon the amount of air at the working face than upon the amount of air which is passing through the shafts.

Air is distributed in mines by the setting up of air currents owing to differences in pressure, etc. The density of the air in one shaft is reduced by either natural or artificial means, while the heavier air descends from the other shaft. The fresh air shaft is called the down-cast, and that by which the impure air ascends, the up-cast shaft. The guiding of this air current is a matter of the greatest moment, and although in theory it appears to be a very simple task, it is not so in practice. Air at high pressure will find its way by every route open to it to places where pressure is low; all that is necessary then to make it take a particular route is to close all the other passages, and it is here where serious leakages and deflections of the air current take place. Here let us repeat that the point, never to be lost sight of, is not that sufficient air is found at the intake, but that sufficient air is being carried to the men working at the face. In some cases it has been found that when the air was measured in the intake and return air-ways, a large and sufficient amount was found, while the amount reaching the men at the face was deficient. In order to guide the air, a screen of canvas or brattice on a frame is put across any road where the current is not desired, these doors being hung so as to close by their own weight. A permanent stopping may be made of wood or brick or other material. Near the working face the screens are generally of canvas or brattice cloth, as the air-currents there are not so strong.

With regard to the position of the ventilating fan, the 1909 *Commission* recommends that, "Fans used for producing the main ventilation of a mine should be placed on the surface, but the use of auxiliary fans underground should not be prohibited. Precautions

must be taken to prevent short-circuiting of the air current where auxiliary fans are used." It should also be remarked that since in explosions the fan houses or the fan itself is often damaged, care should be taken to protect it. Dr Haldane has drawn attention to another very important practical point, still, however, under discussion, namely, the value of reversing the air-current, and the means of doing this: and the Commission draws attention to the importance of collieries being provided with a means of reversing the air current in the event of a fire or an explosion.[1]

The Formation of Gases in Collieries and their Action on the Men breathing them.

In the old days, before much was known amongst miners at least about the composition of air, various kinds of atmospheres which had different actions on a flame, or on the men breathing them, were spoken of as different kinds of "damp." Fire-damp for example, gave rise to a fiery or inflammable atmosphere, hence its name; another which extinguished lights and gave rise to choking sensations when breathed, was therefore known as choke-damp or black-damp; while carbon monoxide was called sweet-damp, probably because of the sweet taste it produces in the mouth, an effect to which we shall draw attention later when discussing poisoning by blast-furnace gas; and, lastly, the poisonous air in mines produced by explosions was and is still known as after-damp, while the poisonous fumes met with in underground fires are called white-damp.

Carbonic Acid (Carbonic Dioxide: CO_2).

If the air in the return air-ways of a pit is examined, it will be found that there may be as much as eight to ten times more CO_2 present than in the air entering the mine. A considerable percentage of this comes from the respiration of men and ponies, from the burning of lamps, etc., and from the use of explosives; but by far the greatest amount probably is given off from the coal itself, as wherever there is oxidation of carbon going on, as at the working face, CO_2 is produced. The oxidation of iron pyrites, which is present in varying amount in different seams of coal, leads to the formation of H_2SO_4 which acts upon the carbonates, liberating CO_2. The percentage of CO_2 present is, as we shall see, greatly increased after explosions, or where there is an underground fire. It has been found that, unlike fire-damp, the production of CO_2 in mines is generally much more uniform and equal, and consequently that examination of the air in the return air-ways generally gives a very good

[1] Royal Commission Report, 1909, p. 207, p. 78-80.

idea of the efficiency of the ventilation. This is not always the case, however; for sometimes most unexpectedly sudden outbursts of CO_2 take place when the gas simply flows out from the worked coal. In some coal seams extraordinarily violent outbursts of CO_2 have been observed. For example, while a mine in the Gard coal-field in France was being developed, there were frequent outbursts of CO_2; indeed there was a continuous discharge of this gas from the seams while boring and other operations were in progress. In 1908, when sinking deeper in the same pit, continuous blasting caused a sudden outburst of gas carrying with it volumes of dust which burst from the shaft like a cloud and rose to a height of 120 ft. This CO_2-laden dust continued to belch forth from the shaft for one and a half hours, with the result that there was a bed of dust about 40 inches in depth deposited on the floor of the winding engine-house, and the ground all round the pit-head, covering fifty or sixty acres in extent, was black with coal-dust. Three workmen then at the pit-head were unable to escape and were asphyxiated; people living in the cottages in the neighbourhood of the pit-head were partially asphyxiated; and many small animals, cats, dogs, fowls and birds in the neighbourhood were afterwards found dead. In 1896, at Rochbelle in France, twenty-four men were asphyxiated owing to a sudden outburst of CO_2. Fortunately, however, such accidents are extremely rare.

Carbonic acid gas is a narcotic poison. It has a direct poisonous action on the organism, but it must be present in very large quantities before it threatens life. When a man breathes a large percentage of this gas, or when in a chamber or caisson he breathes it in a compressed state, it produces its poisonous action, because, owing to the high CO_2 tension in the air, it prevents the blood giving off CO_2. It is therefore directly poisonous. At the same time, there is the action produced by the deficiency of oxygen in the air, owing to the increase of CO_2.

It is well here to draw particular attention to the fact that the breathing of large percentages of CO_2 or of CO in compressed air may have very dangerous after-effects. When dealing with carbon monoxide we shall have reason to insist on this danger. *Sir Thomas Oliver* [1] found when mice were exposed for half an hour or more to air containing 1 per cent. CO_2 compressed at five atmospheres that on being taken out of the chamber they quickly recovered, although at first they were sluggish in their movements and inclined to sleep. A certain number of them, however, died a few days afterwards (in some cases as long as ten to twelve days afterwards) from inflammation of the lungs, while in other cases death took place suddenly a few hours after they were removed from the caisson. The fact of carbon monoxide acting as a direct poison on the nerve centres and causing sudden death will also be discussed later. Excess of CO_2 in compressed air is much more dangerous than

[1] Thomas Oliver: *Diseases of Occupation*, p. 57.

even a large percentage at ordinary atmospheric pressure. It is seldom, however, that CO_2 is present unaccompanied by other gases, such as CO as in after-damp, in quantity sufficient to cause death. Where it is present in considerable quantity and where exposure to it causes death, the fatal termination is generally the result of the relatively small percentage of oxygen present in the atmosphere breathed.

Large quantities of this gas are present in old workings, and may be met with in sinking operations or in opening up old shafts, but in these circumstances it is generally the deficiency of oxygen which is the serious factor, and not the increased percentage of CO_2.

Leblanc,[1] who carried out a large number of experiments on animals confined in air-tight compartments, found that he could shut up a dog in an atmosphere containing 30 per cent. CO_2 for three quarters of an hour, and that the animal recovered very quickly. *Snow*[2] found that in an atmosphere containing 20 per cent. CO_2 a white mouse died in an hour and a half, that in 12 per cent. CO_2 (oxygen being normal) a sparrow died in two and a half hours, while a white mouse recovered after remaining twelve hours. *Guerard*[3] in 1843 also demonstrated by experiments that large quantities of CO_2 could be inhaled with impunity; and *Brown-Sequard*,[4] in his experiments to show that toxicity of expired air does not depend on CO_2, was able to breathe 20 per cent. CO_2 without being much affected. He also exposed dogs to 45 per cent. and 50 per cent CO_2 for more than twelve minutes without causing death.

Symptoms of Carbonic Acid Gas Poisoning.—As regards the amount of CO_2 present which may give rise to symptoms of poisoning, *Haldane and Lorraine Smith*[5] carried out a number of experiments on animals and men, and demonstrated that these symptoms did not begin to appear till from 3 to 4 per cent. of the gas was present, when the breathing became slightly affected. Men, however, can go on working for a considerable time in this atmosphere without feeling serious discomfort, although they will certainly become more quickly fatigued, and great exertion will cause panting; but Haldane kept animals for weeks in this atmosphere without causing them much inconvenience. This fact has been recognised for many years in the mining world, and *Thomas*,[6] who wrote the first scientific work on mine gases, found that working in the dip in certain coal seams in South Wales where, owing to the non-completion of air-splits, the ventilation was very bad, men would often work for hours together in an atmosphere containing 2 to 5 per cent. CO_2.

Haldane and Lorraine Smith found that where the air becomes worse,

[1] Leblanc: *Annales de Chimie et de Physique*, 1842, p. 223.
[2] Snow: *Lancet*, 6th April, 1839, p. 93.
[3] Guerard: *Annales d'Hygiène Publique*, 1843, II., p. 55.
[4] Brown-Sequard and d'Arsonval: *Compt. Rend. de l'Acad. des Sc.*, 1889, p. 267.
[5] Haldane and Lorraine Smith: *The Journ. of Path. and Bact.*, 1892, I., p. 174.
[6] Thomas: *Coal, Mine-Gases and Ventilation*, 1878, p. 146.

the symptoms become more marked, and when 6 per cent. is present there is distinct breathlessness and panting, palpitation of the heart, with the pulse faster and of higher tension and bounding, and slight frontal headache. A percentage of 7 to 8 per cent. causes more urgent symptoms, while with 10 per cent. the distress is very great, the headache becomes much more severe, there is marked dyspnœa and throbbing and flushing of the face, and the gas begins to have a stupefying effect. With 12 to 15 per cent. cerebral symptoms appear, and the patient soon becomes unconscious. But even although the patient has been lying unconscious in such an atmosphere for some time, he may recover—the unconsciousness thus induced by CO_2 being seemingly much less dangerous than that brought about by a deficiency of oxygen. Death may take place after exposure for several hours to 25 per cent., but Haldane found that a much greater percentage, even 50 per cent., may in the case of some animals be breathed without causing death. In experiments made by *Wilson*[1], a rabbit was placed in a mixture of 10 per cent. CO_2 and 90 per cent. atmospheric air for an hour and seven minutes. The only effect seemed to be exhilaration; for, on being released, this rabbit appeared livelier than the others amongst whom it was placed. After exposure for an hour to an atmosphere containing 25 per cent., the rabbit was as lively as ever; but when the amount of CO_2 was increased to 50 per cent., it began to gasp in two minutes and died in seventeen minutes. In an atmosphere of 75 per cent. death took place in 10 minutes. Experiments with mice led Wilson to conclude that small animals are less resistant than the larger to the effects of CO_2. With regard to the *action of CO_2 on flame*, Wilson also found that the presence of 16 per cent. CO_2 extinguishes the ordinary safety-lamp flame; therefore respiration may go on unimpaired in air containing at least 10 per cent. more CO_2 than is required to extinguish flame.

Haldane and others have pointed out that breathlessness is produced much more quickly by excess of CO_2 than by deficiency of oxygen. The important practical point to remember, therefore, is that breathlessness caused by CO_2 starts long before there is any serious danger, while when it is met with as the result of deficiency of oxygen, or in CO poisoning, where, of course, there is also deficiency of oxygen, it is a grave symptom and points urgently to serious danger.

Black-damp. (Choke-damp.)

The most common atmospheric impurity met with in collieries is black-damp. No pit is entirely free from a certain proportion of it, and practically every experienced miner has seen its action on the flame of his lamp. That is how he recognises its presence. Black-damp is

[1] Joseph R. Wilson: *American Jour. of Pharmacy*, 1894, Vol., LXV., No. 12.

now regarded as the residual gas which remains after the action of the oxygen of the air on oxidisable matter present in coal. Haldane was the first to show that pure black-damp free from air is a mixture of nitrogen (87 per cent.), with a relatively small proportion of CO_2 of 13 per cent.; but as generally found in collieries, it may be regarded as a mixture of air and black-damp, that is to say, there is a decrease in the oxygen percentage and an increase in the carbon dioxide. In certain cases it has been found, however, that although the quality of the air was sufficient to immediately extinguish lamps, and although it could not support respiration for any length of time without producing serious effects, yet on analysis the sample showed only a very small percentage of CO_2, the dangerous factor being most probably the marked diminution in the amount of oxygen. *Bertram Blount*[1] reports the remarkable instance of a sample of choke-damp being found which was quite free from CO_2. The sample was taken from a room used for cold storage where the temperature was about 20°F. The reason attention was directed to the atmosphere was that candles refused to burn in it; usually electric lights were used. Men doing slight manual work in it complained of a slight feeling of oppression. A sample was proved on analysis to contain 17·6 per cent. of oxygen and 82·4 per cent. of nitrogen, the amount of CO_2 being negligible, not amounting to more than 0·1 per cent. The explanation of the oxygen-poverty of the air was that a shaft from a disused well opened into the room, from which large volumes of this air were discharged. The air of the well was analysed and found to contain oxygen 8·7 per cent. and nitrogen 91·3 per cent.

An average taken from samples of black-damp procured from many coalfields will give from 10 to 16 per cent. of CO_2. The recent analyses of the air in certain mines carried out by Prof. Cadman demonstrated that the percentage of CO_2 in black-damp varies from 3 to 21 per cent; generally the percentage of CO_2 in the mixture is from 10 to 12 per cent.

As black-damp usually has a greater density than air, it is to be found on the floor and in low-lying workings. It happens occasionally that the gas is present in a heavy stratum below, with the lighter air above, and these strata are so sharply defined that the lowering of a lighted candle but one inch below a certain level would immediately extinguish it. In compensation cases in Court it will probably be found that a great deal of stress is laid on the condition of the flame of the miner's lamp, and that as long as the flame burns, no matter how dimly, the air is believed to have no poisonous effects. But we often find in these cases that the lamp has been hung well up in the roof, and that the man at the face, lying as he does on the pavement or floor, may be working in very much worse air, whereas were the lamp placed on the pavement it would in all likelihood be immediately extinguished.

[1] Bertram Blount: *Journal of Hygiene*, 1906, p. 175.

It is well known that black-damp is often met with in great quantity in all shallow mines where a new seam or section is opened out; indeed, in some cases, it seems simply to pour out of the freshly-cut coal. When a place has been empty for some time and has not been worked, it is very often found that it is filled with black-damp and that there is a very small percentage of oxygen present, that gas having been used up in uniting with the oxidisable matters in the coal. Considerable quantities of black-damp may collect in places which have been recently declared free and good by the fireman, owing to differences in atmospheric pressure. This is especially the case in shallow mines. Owing to its relatively high specific gravity it is generally found in considerable quantity in dip workings, and it accumulates at the lower levels, which is a most important point in the ventilating of a pit, as it is sometimes very difficult to deal with.

Since black-damp is generally heavier than air, and consequently collects about the floors, the result is that when men are knocked over and rendered unconscious, or when they become sleepy, as they sometimes do when the percentage of oxygen present is small and large quantities of CO_2 are also present, their condition will be rendered worse by the heavy gas collecting about the pavement. If they are not removed at once their condition may become serious. In old and unventilated workings, and at the bottom of sumps, in re-opening old shafts, etc., the greatest care should be taken.

The work recently done by Cadman shows that the specific gravity of black-damp varies considerably in certain mixtures. It may frequently be regarded as not more and sometimes much less than that of air, as for example, when the black-damp contains less than 5·25 per cent. CO_2 and where fire-damp is present, the combined gases may be lighter than air. Cadman lays great stress on the importance of this, and points out that the greatest caution should be exercised where any accumulation of black-damp is found of lighter density than air, especially in mines worked with naked lights, as this lesser density is probably due to the presence of fire-damp.

Black-damp differs from fire-damp in being non-explosive, but when in combination with the latter, as it sometimes is, the fire-damp may cause explosions. Miners generally gauge the amount of black-damp present by its action on the flame of their lamps, which burns with decreasing brilliancy as the percentage of CO_2 increases, and is extinguished if more than 10 to 17 per cent. of CO_2 is present. This action greatly depends, however, upon the amount of oxygen present, which may vary considerably. The effect on the flame is now regarded as chiefly due to the deficiency of oxygen. If a lamp burns well, it indicates that there is more than 17 per cent. of oxygen present, and therefore that the symptoms of poisoning could not be caused by the deficiency of the

oxygen. *Clowes*,[1] from his experiments, found that the oil flame was extinguished in 30 seconds by air containing 16 per cent. CO_2, while it required 58 per cent. CO_2 to extinguish the hydrogen flame in the same time. As one can work in much higher percentages of CO_2 than is required to extinguish the flame of an oil lamp, the knowledge of this fact about the hydrogen flame is useful. Clowes found that 26 per cent. was the minimum at which breathing was affected.

The Royal Commission of 1909 in their Report found, as regards black-damp, that the standard of ventilation should be such that a lamp or candle will not burn dimly, or any appreciable effect be produced on the breathing of the men employed. Air found by analysis to contain less than 19 per cent. of oxygen, or more than 1·25 per cent. of CO_2 should be regarded as below the standard.

Symptoms of Poisoning by Black-damp.—Haldane and Atkinson[2] found when 26 to 27 per cent. of black-damp is present that symptoms of poisoning begin, the breathing becoming deeper and more frequent as the lungs make an effort to dilute the amount of CO_2 in the lung air to the usual proportion (about 5½ per cent.). As the percentage increases the breathing becomes more affected, till with large percentages it becomes markedly embarrassed. With 50 per cent. there is violent panting, throbbing at the temples, great frontal headache, and flushing of the face. The flush on the face deepens and becomes bluish; when cerebral symptoms commence (with 60 per cent.) the tint becomes leaden. The cerebral symptoms, which have now set in, exhibit themselves as loss of power over the limbs, staggering gait, giddiness, and dulness of cerebration, and these are due chiefly to the want of oxygen.

Now the question is:—To what are these effects to be attributed? Is it to the presence of CO_2 or to the want of oxygen? Haldane has drawn up a table showing the effects of analysed airs on persons breathing them, and he finds that symptoms start when the percentage of CO_2 rises to 3½ per cent. The oxygen in this sample had fallen to about 15 per cent. which would not affect breathing, therefore the CO_2 is the poison. He thinks that the symptoms already described, excluding blueness of the face, are due to CO_2, and after that to the deficiency of oxygen. The danger signals from the presence of CO_2 are thrown out far in advance, while with CO or marked deficiency of oxygen there may be no such warning. In very bad air, then, the amount of CO_2 would give warning of the next and more serious danger, the want of oxygen.

[1] Clowes and Redwood: *Detection of Inflammable Gas and Vapour*, 1896, p. 162.

[2] Haldane and Atkinson: *Investigations on the Composition, Occurrence, and Properties of Black-damp.* Trans. Inst. M. E., Vol. VIII., 1894, p. 561.

TABLE I.[1]

Component Gases.	Points at which breathing became noticeably deeper.		Points at which Panting was very severe.		
	Nabb Pit, Lillieshall Colliery.	No. 4 Pit, Podmore Hall Colliery.	Nabb Pit, Lillieshall Colliery.	No. 4 Pit, Podmore Hall Colliery.	Dip in Forge Pit, Apedale.
Oxygen	15·30	14·84	9·60	10·11	10·08
Nitrogen	81·32	79·26	83·08	79·72	82·30
CO_2	3·38	3·26	7·32	6·01	7·62
CH_4	0·00	2·64	0·00	4·16	0·00
	100·00	100·00	100·00	100·00	100·00
Percentage of Black-Damp	26·79	26·36	54·07	47·47	51·77

Doctors in colliery practices have frequently to deal with men who, after working in bad air for some time, complain of headache, giddiness, ringing and noises in the ears, and sickness. Where the air is very bad, the men while at work will complain of breathlessness with oppression and constriction about the chest, palpitation with uneasiness, sometimes a severe pain at the heart, and a feeling of being utterly worn out. The men have to take frequent rests, and they find their work is very much heavier than usual; for example, one man informed us that he took seven hours to do some work he should have finished in less than two hours. The lightest work requires great exertion. In some cases giddiness is a marked symptom. In one of our cases, this symptom lasted for thirty-six hours, persisting even when the man lay down in bed and shut his eyes, and with his eyes closed he felt the bed rocking. In the same patient there was a throbbing pain in the right side of the head and ringing in the ear, confined to the right ear. The headache in nearly all these cases is severe. It is made worse by stooping; indeed becomes almost intolerable with any exertion; and it is generally this symptom, with the giddiness which usually develops, which compels the men to stop work. One man described the pain in his head when he stooped as if the front of his head was about to drop out. On the way home from work the throbbing pain in the head and the giddiness may bring on vomiting. We have had cases where the vomiting lasted more or less for four days. It may be accompanied by severe pain in the epigastrium. Where there is no pain, the patient frequently complains of nausea. In

[1] Haldane and Atkinson: Trans. Inst. M.E., Vol. VIII.

other cases a very severe attack of diarrhœa follows, accompanied by severe pain in the abdomen. This last symptom is very much more frequently met with in poisoning by black-damp than in CO poisoning. Sometimes it may be very severe, and in spite of all treatment may last about a week.

In nearly all severe cases the muscular power of the legs is affected, the legs sometimes, indeed, becoming almost quite powerless. The patient staggers about and can hardly walk. Although most of the symptoms become less marked when the patient gets out to the open air, the exertion of walking makes the staggering gait more pronounced, and generally increases the pain in the head. Patients also declare that the lamp which they carry on their caps feels extraordinarily heavy, as if almost to weigh them down. The pătient may become very sleepy, and complains then of a gradual loss of power which may develop to complete loss of consciousness. A very common symptom which comes on after the headache wears off is drowsiness and sleepiness ; wives frequently offer the statement that when their husbands or sons are working among bad air they fall asleep when they come home in the afternoon, and that they are very difficult to rouse in the morning.

The symptoms here mentioned are obviously those of gradual asphyxiation, the poisonous effects of the CO_2 being intensified by the fact that there is often a deficiency of oxygen present. The result is that the men who are doing the hardest muscular work are the first to suffer, and they suffer most severely. This is a fact observed by ourselves and one which has been insisted upon by one or two other observers. Severe muscular exertion in a polluted atmosphere with increase of CO_2, together with a diminished percentage of oxygen, quickly causes severe symptoms of poisoning to appear, and if work is persisted in after the appearance of these, unconsciousness and even death may ensue. Where two or three miners are working together in very bad air, it is found that the man who is working at the face (working as he does on the pavement in the worst air, and beside the freshly-cut coal from which the gas is constantly exuding) suffers very much more than his companions who are "drawing," and who consequently get periodically into better air as they push along the hutches.

Where black-damp contains a small percentage of CO_2 and a very large percentage of nitrogen with very little oxygen, it must be borne in mind that a person exposed thereto may collapse into unconsciousness with practically no warning. Sometimes the men will say, when a well-defined cap forms on their lamps, that the symptoms were caused by the presence of "gas" (fire-damp), but in these cases it will be found that it is the black-damp and not the fire-damp which is the poison.

Haldane, Leonard Hill, and others have called attention to the important bearing the temperature has in regard to the action of

poisonous gases in mines. With regard to the percentage of CO_2 it is found that where there is a good circulation of cool air by fan-action, this may be raised considerably without causing any discomfort to the men, but if the fans are put off and the temperature consequently rises, the men at once begin to feel serious effects.

These poisoning cases are seldom seen among miners till their arrival home, and most of them make rapid recovery with practically no further treatment than rest in bed and a dose of salts and cream of tartar in the morning. This is the favourite cure, as the men believe that "it thoroughly cleanses the blood of the gas." The other part of the cure is a good stiff dose of whisky as soon as the men are able to procure it. There can be no question, in cases where men have been working in badly ventilated places and have been overcome, that when they reach the surface a stimulant, the favourite being whisky, quickly brings them round; the good result being probably due to the action of the alcohol on the respiration and circulation, accelerating and increasing their activity and so helping to get rid of the CO_2 while at the same time increasing the supply of oxygen to the blood. After a dose, or as the miners say, a "feed of damp," a man may be "off his usual" for a time, be very drowsy and drop off to sleep at all hours and in any position, feel disinclined for work, and complain of headache and indigestion with loss of appetite, but usually, as has been already said, these symptoms rapidly pass off. In other cases, however, the victims are not so fortunate, and we have seen cases where certain of the symptoms persisted for a few months. In these cases the symptoms complained of are breathlessness on exertion, oppression about the chest, palpitation and pain about the region of the heart, with tachycardia, frequent headaches, giddiness on exertion, being easily fatigued, and, when tired, of loss of power in the legs with staggering gait. In these cases no cardiac lesion nor dilatation could be detected, but the heart sounds were weaker. Cases of acute bronchitis and pneumonia have occasionally been met with after poisoning by black-damp.

Men who habitually work in bad air, but which is not sufficiently tainted to cause the acute symptoms which have been mentioned, may suffer from slight anæmia, with disinclination for work, breathlessness on exertion, lassitude, feeling of "not being themselves," loss of appetite, and other symptoms of indigestion. Return to work in better air, however, soon puts them right, and we have never seen any serious permanent symptoms following prolonged exposure to black-damp. But we can quite understand that men who habitually work in a mine where the ventilation is very bad and who have excessive muscular work, may develop hypertrophy of the heart and other cardiac troubles owing to the greater strain on that organ.

Attention is now directed to a matter which is not infrequently

cropping up in workmen's compensation cases in Court, viz. :—the effect which working in bad air has upon those who suffer from heart lesions. One such case is before us at the present time; that of a man with heart trouble who continued to work a small fan in a collection of black-damp in order to dissipate it, notwithstanding that his lamp was out, and who afterwards collapsed, suffering from all the signs and symptoms of acute cardiac dilatation. No man with heart disease should be permitted to work where black-damp is present in such proportion as to affect the lamps; for such a man the strain of working hard in an atmosphere deficient in oxygen and with a high percentage of CO_2, is quite as bad as the running of a hundred yards would be. Those suffering from chronic bronchitis and phthisis stand working in impure air very badly; —in two cases of phthisis, where the men continued working in very bad air as long as they could, the disease ran a particularly rapid course.

Some degree of toleration may be established among miners working continually in bad air. Newcomers to a section where the air is very bad, and in which others have worked with impunity for some time, may be compelled to give in. It may be in these cases that the new men find themselves much more quickly fatigued with their work than they would were they working in better air, and perhaps, in addition, they find that they are unable to do the same work as those in whom toleration has been established. For example, when new seams are being opened up or new headings driven into the fresh coal, men who have been working there for a long time may not complain at all, although where there is no through ventilation the air must be very bad, containing a large proportion of CO_2 and a lessened quantity of oxygen. A newcomer to such a section may in a very short time be overcome. Again, some men declare themselves immune against bad air; and, indeed, it would appear as if these are able to work in air which few others would care to face. Cases are on record of a man, going to the rescue of his mate who had been overcome by black-damp, being himself overcome, and when both had been brought to safety, it was found that the rescuer was dead, while the other, who had been longer in the poisonous atmosphere, recovered. It has been again and again demonstrated, in removing the rails from abandoned workings in which so much black-damp had collected that electric torches had to be used, that in a gang of, say, eight men doing the work, one might be very rapidly overcome while the others remained working for a considerable time with only a little discomfort.

Miners are apt, inappropriately, to lay many troubles at the door of bad air; such as indigestion, tightness about the chest, palpitation and breathlessness on exertion, and, perhaps, even some major complaints which could not possibly have been caused by "bad air." In one instance, for example, a man who developed epilepsy, declared that it

was the result of working continuously for two or three years immediately before its onset in badly-ventilated places which were frequently tainted with gases of combustion from shot-firing, although he could not explain away very well the fact of there being an epileptic taint in his family.

In the opening up of old shafts and old workings, etc., there is always risk of gas-poisoning. In these cases, it is undoubtedly the serious deficiency in the amount of oxygen present which is the danger—not so much the high percentage of CO_2. If it were the latter, the men would receive early intimation of the serious state of the atmosphere by various symptoms. But the action of the poisonous atmosphere in many of these cases is very rapid; indeed the men may drop before they know that anything is wrong. In a certain district, in going down an old shaft which had been partly filled up and which was being opened again, a manager and one of his men were immediately overcome and died before assistance could be procured. Such accidents, in fact, in old workings are frequent. Quite recently (Sept. 1910) two young men were exploring some old colliery workings near Wigan when they were overcome. A third youth observing that something was wrong, gave the alarm, and the father of one of the youths, in attempting rescue, was also overcome, but he was dragged out with a rope. Both young men were dead when got out.

One cannot leave this subject without referring to the excellent work of Haldane on choke-damp in wells. In nearly all text-books on Medical Jurisprudence the active gaseous poison has hitherto been regarded in all these cases as CO_2. But Haldane has pointed out how rapidly the poison has acted in all such cases of gas poisoning in wells, etc., there being hardly any appreciable premonitory symptoms before the occurrence of loss of power over the limbs and giddiness. Consequently, if a man were going down such a well by means of a ladder, his legs might give way, and he would probably fall before he realised his danger. The lesson therefore is, that the greatest possible care should be taken in all such cases (going down wells, disused shafts, etc.), to guard against such accidents. Prevention is after all best; that is to say, means should always be taken to ascertain the condition of the atmosphere before a descent is made, and even if the air is supposed to be right, care should be taken to fasten a rope to the person making the first descent. Many of the ills, then, which were formerly set down to the action of carbonic acid must now be recognised as caused by a deficiency of oxygen in the air.

Fire-Damp (Methane, Marsh Gas, Carburetted Hydrogen, CH_4).

Fire-damp consists principally of marsh gas mixed with varying proportions of CO_2, nitrogen, and oxygen. Its specific gravity is generally

about 0·5, or it may be as high as 0·7 or 0·8 when large proportions of other gases are present. It is sometimes mixed with some other heavier hydrocarbons, *e.g. ethane.* It is generally found that CO_2 is present also in greater or less amount; for example, in old workings it has been proved that in course of time there is a gradual decrease of CH_4, and increase of CO_2. Fire-damp is lighter than air, and consequently ascends to the higher workings, where it is often found in greater quantity under the roof, accumulating in cavities formed where falls of stone have occurred, especially where the ventilating current is slow, rather than in the main air shafts where the gas becomes diffused and diluted.

Pure marsh gas has no smell, but fire-damp, as found in mines, has generally an appreciable odour; indeed, miners can often detect its presence by the smell, which has been compared by some to onions.

Methane has no poisonous action on man, and one might breathe with impunity an atmosphere containing a large percentage of methane, provided the atmosphere contains a sufficient amount of oxygen. So long as the amount of oxygen is not seriously reduced, no ill effects can result from the breathing of fire-damp. As Paul Bert pointed out long ago, the physiological effects of fire-damp are just like those produced by air from which a proportion of its oxygen has been removed. Contrary to the general opinion, however, that fire-damp produces no effects unless CO or CO_2 is present, or where the percentage of oxygen is much lowered, *Lussem*[1] has demonstrated by experiments that where a large percentage of CH_4 is present, a stupefying effect is produced. For example, he found that when animals breathed a large quantity, they became very sleepy, while on men also it had a stupefying effect. But in all cases recovery was very rapid when the patient was placed in fresh air. Experimenting with olefiant gas, he found that this action was much more pronounced, as it had a distinct narcotic effect on man.

It is well to remember, however, that the very presence of these different gases in the air means a reduction in the amount of oxygen present, and also that wherever there is much methane, it necessarily follows that there is also present an increased amount of CO_2. In itself then, marsh gas has no toxic action on man except in the absence of sufficient oxygen, when it may lead to asphyxiation. We adduce a few examples of the ways in which fire-damp accidents are produced. In the year 1911, in a pit at Croy, owing to gas collecting in a place after blasting (the result of blasting, we should add, frequently allows collections of fire-damp to escape through fissures and falls, etc.,) the men were prohibited from working. A few days afterwards the fireman heard moaning in this closed place, and going up the slope, he found two men, one dead and the other unconscious. In April 1911, at Loanend

[1] Lussem: *Experimentelle Studien über die Vergiftung durch Kohlenoxyd, Methan und Æthylen.* Inaug. Dissert., Berlin, 1886, p. 19.

Colliery, Cambuslang, the under-manager had proceeded through old workings with a quantity of cloth to stave off an accumulation of gas, when he was overcome by fire-damp. Repeated efforts by several men, including the manager, to reach him were made, but all of them were in turn stupefied by the fire-damp, the manager being so seriously overcome that it took a long time to bring him round. When the underground manager was taken out he was found to be dead. A number of fatal cases of poisoning by fire-damp, quite distinct from black or choke-damp and after-damp, are reported in the *Prussian Fire-damp Commission's Report*.[1] From 1861 it was found that in Prussia fifty-four cases of suffocation by fire-damp alone occurred. These occurred principally in three coal basins, viz., Lower Rhine, Westphalia, Aachen, and Saarbrück, forty-eight of the fifty-four occurring in the "rise" working places, one in an old working, three at the top of inclined planes, one in a working place, and one in a "rising" shaft. In the case which occurred at the "face," it was due to a sudden outpouring of fire-damp, whereas in all the other cases it was due to accumulations of gas where the place had been idle; in other cases it was owing to insufficient ventilation. In twenty-six of the cases that occurred in the "rise" working places, the men had been gradually overcome while at work.

It is not unlikely, however, that many of the cases attributed to fire-damp which occur at the face in badly-ventilated places, are due to black-damp or de-oxygenated air. Amongst miners there is very great confusion regarding the nomenclature of gases.

Besides its direct action, CH_4 has another and most important action which is of the greatest interest. What has made this gas one of the most deadly enemies of the miner is the fact that mixtures of it with certain proportions of air are inflammable, and when ignited, lead to most disastrous explosions. In itself CH_4 is non-explosive, and will not support combustion. If one volume of fire-damp be mixed with four volumes of air, the mixture will burn feebly; if with 5·5 volumes it will explode feebly, but every added volume of air after this figure increases its explosive power till the maximum is reached, viz. :—9·5 volumes of air to one of gas; in other words, when perfect combustion of methane has been obtained in that proportional admixture. The chemical equationof the reaction is as follows :—

$$CH_4 + 2O_2 = CO_2 + 2H_2O.$$

And since one cubic foot of air contains 0·21 cub. foot of oxygen, therefore 9·5 cub. feet of air will contain 2 cub. feet of oxygen. Beyond this proportion, the addition of air lessens the explosive action. With fourteen volumes of air (7 per cent. gas), the force of the explosion is very much reduced, while with thirty vols., of air (3·3 per cent. of gas) the mixture is no longer explosive. We hasten to add, at the same time, that these

[1] *Prussian Fire-damp Commission Report*, pp. 19 and 20.

figures apply to mixtures of fire-damp and air only, for it is now recognised that where coal-dust is present, very much smaller quantities of fire-damp may lead to explosions; indeed, 1 to 2 per cent. of gas may under these circumstances constitute an explosive mixture; and the presence of other members of this group may also have the action of causing small percentages of gas to have an explosive power.

Fire-damp is most commonly found in deep mines, as in shallow mines it generally manages to find its way to the surface through fissures in the strata, although coal itself is permeable and gas may stream through strata many feet thick (thirty to forty feet). Mines in which fire-damp is present in large amounts are termed by the miners "fiery." Mines differ greatly in this quality, as some coal seams give off large quantities of fire-damp, while others are practically free from it, or the gas may only be met with at long intervals. In Lanarkshire, for example, it is found that within a very short radius some pits may be "fiery" while others are practically free from gas. Some seams offer a much better opportunity for the gradual escape of the gas almost as it forms, while in others, owing to the conformation of the strata, it may be shut up. Thus it is that in a colliery which has never previously been troubled with gas, a large quantity of fire-damp may suddenly appear, owing to the tapping of a seam containing a large quantity of confined gas. Occasionally in some seams when such pent-up gas is tapped, large quantities of gas may suddenly burst out with great force, and these *sudden outbursts of gas* were formerly regarded as the causes of nearly every large colliery explosion. "Blowers" are formed by gas streaming from fissures continuously and in large quantities. Where the pit roads pass near or through faults, fire-damp is often given off in large amounts; and when this happens, a so-called safe mine may in a few moments be rendered very dangerous. Very violent outbursts of fire-damp, just as outbursts of CO_2 and black-damp, may take place and lead to serious disasters. In some coalfields in Belgium, for example, sudden outbursts of enormous quantities of fire-damp occur without the slightest warning, rush through the pit, and even ascend to the surface to take fire there: when this happens the effect is appalling.

The most serious eruption of fire-damp, perhaps, which has ever taken place, occurred in L'Agrappe Colliery in Belgium in 1879, in which 121 men lost their lives and many were injured. It was calculated that in three hours 12,000,000 cubic feet of gas escaped. This ascended the shaft and caught fire at the pit-head, destroying all the buildings in the vicinity. The flames were extinguished by an explosion which was repeated five times at intervals of ten minutes. The last explosion, which was the most serious, took place after a quiet interval of nearly an hour. Old workings and goaves often contain large quantities of this gas, which may find its way into the roads, and consequently it has been

found necessary in some fiery mines, where it has collected in large quantities, to shut off the old workings altogether. Extensive blasting operations at the face may liberate large quantities; and sudden outbursts of enormous quantities of gas have resulted from the opening up, either by cutting through into or by falls of the roof of a natural cavity in the coal. A remarkable accident happened lately in Culross, Fife, which led to the death of three miners. In this case it was supposed that there had been a large collection of gas in a cavity, the outside of which had become so thin that it was unable to resist the force of the enclosed gas which burst through with such terrible force that it dislodged about 100 tons of coal and other material, shattering these almost to powder. The extent of the fall was about twenty yards. One of the bodies of the victims was found buried under the débris; the other two men appeared to have been suffocated by the dust and CH_4.

Explosions are frequently the result of accidental ignition of accumulations of fire-damp. The gas may be ignited by a naked light where, for example, a pit is supposed to be free from fire-damp. It has been pointed out very often that a large number of explosions take place not in the fiery mines, but in those regarded as "safe." With the present recognition of the dangers of coal-dust as an explosive agent, no mine should be regarded as immune from explosions. An explosion may be set up sometimes by the striking of a match to light a lamp which has gone out (safety locks, where in use, do away with this evil), or to light a pipe; of course naked lights, the carrying of matches, etc., are forbidden in fiery pits, although regulations and punishment for contravening these are not always successful in instilling caution. A defective safety lamp may also be the means of igniting the gas. One of the most common causes is the firing of shots; many of the serious explosions which have had their origin in a local explosion of fire-damp and have been propagated by coal-dust, have arisen in this way.

If fire-damp be present in a mine and a lighted safety lamp brought into it, it will be found that the combustible gases present become ignited and burn when they come in contact with the flame, forming thereon a cap of blue flame. To detect this cap, and know from its size and appearance the amount of fire-damp present, requires considerable experience, practice, and skill, and to detect very small percentages of this gas certainly requires much more than the average fireman possesses.

Sir Simeon Snell[1] has recently drawn attention to the fact that it is impossible for a fireman who has nystagmus to gauge a firecap properly, even if he could detect it with normal vision, which many of them cannot. Regarding this visual test for fire-damp, recent investigations, carried out for the Royal Commission, point to the important fact that

[1] Sir Simeon Snell: Presidential Address to Brit. Med. Assoc., *B.M.J.*, August 1908.

many miners are unable to gauge the presence of fire-damp where the percentage present is much under 3 per cent. In order to detect the cap, it is necessary to lower the flame in the lamp until a small, non-luminous flame is obtained. By a skilled observer, as little as 1 per cent. of fire-damp can thus be detected; with 2 per cent. the tip of the cap becomes visible, and a definite and distinct cap, a quarter of an inch high, may be seen. With higher percentages the cap increases in height and becomes denser in colour; with 3 per cent. it is half an inch high, and with 4 per cent. one inch high. It is now recognised that very small percentages of fire-damp, in the presence of coal-dust, may constitute an explosive mixture. In order to detect fire-damp there are special lamps now in use.

Sulphuretted Hydrogen, H_2S (Stink-damp).

This gas is most poisonous, but happily it is not found in mines in large quantity. It is given off in small quantity in underground fires and after explosions, when probably it causes the smarting of the eyes and throat complained of by those exposed to it. It is also given off at blasting operations with gunpowder, but in negligible quantity. It is formed by the decomposition of organic substances containing sulphur, and from coal as a secondary product of decomposition, being sometimes found in "blowers" of gas, to which it gives a peculiar smell. From experiments carried out by Lehmann and Oliver on men and animals, the terribly poisonous character of H_2S has been demonstrated. *Lehmann*[1] found that exposure for an hour or more to air containing as small a percentage as 0·07 of H_2S may be fatal. In man, serious symptoms were developed within a few minutes of exposure to 0·05 per cent. Also that 0·2 per cent would kill animals, such as dogs, in one or two minutes. *Oliver*[2] found that exposure to 0·15 per cent. suddenly caused the animal to become rigid and lifeless, and that exposure to such an atmosphere even for a few seconds caused gasping and great distress; and often the animal, with a short cry, dropped down dead in a state of great rigidity. Death is probably due to paralysis of the respiratory centre. *Wilson*[3] again found that 0·5 per cent. H_2S killed a rabbit in three minutes, 0·2 per cent. in ten minutes, and 0·1 per cent. in thirty-seven minutes.

In mines, however, the gas is never present in such dangerous quantity; the percentage found in the fumes from gob-fires, for example, never causing anything more serious than smarting of the eyes and throat and irritation of the air passages, and also a feeling of great thirst.

[1] Lehmann: *Archiv für Hygiene*, 1892.
[2] Thomas Oliver: *Diseases of Occupation*, p. 75.
[3] Wilson: *American Jour. of Pharmacy*, Vol. LXV., 1894, No. 12.

Changes in the Air of Mines after Explosions: Formation of After-damp.

After-damp is the name of that deadly mixture of gases which is formed after explosions, and which miners have cause to dread, since it has occasioned from time to time serious losses of life in mines; indeed, one can say that if the formation of this gas by explosions could be prevented, the danger from explosions would be greatly minimised, since it accounts for by far the greatest number of deaths in coal-dust explosions.

When an explosion occurs in some of the ways we shall indicate, that part of the mine-air immediately loses a great part of its oxygen which has united with the CH_4 leaving nitrogen and carbon dioxide; and where an inflammable mixture of fire-damp and air is fired in which there is an excess of fire-damp (that is more than 9·5 per cent. by volume of mixture), carbon monoxide as well as carbon dioxide is formed. For example, the French Fire-damp Commission showed that after combustion of 12 per cent. fire-damp mixtures, and when the generated steam had condensed, the resulting air contained 4·8 per cent. of CO_2, 2·5 per cent. of CH_4, 3·5 per cent. of hydrogen, 3·9 per cent. of carbon monoxide, and 88·2 per cent. of nitrogen respectively. Where less than 9·5 per cent. of CH_4 is present in the air, CO_2 and steam only are produced. *Baumann*,[1] for example, has shown that where not more than 4 per cent. of fire-damp is present, even traces of CO were not found.

Where there is deficiency of air, and the combustion of the carbon is, therefore, incomplete, CO is always produced. We note here that the experiments carried out by *Thomas*[2] and *Meyer*[3] first demonstrated that large quantities of CO were formed during all explosions. It will be seen later, when discussing the part played by coal-dust in explosions, that where there is much coal-dust present in the mine, a considerable percentage of CO is formed. This is found especially if the mine is badly ventilated, as may happen when there has been a series of explosions occurring in succession, and where these have brought down large portions of the roof and so interfered with ventilation, because, owing to deficiency of air, combustion is incomplete. This, in addition to the deficiency of oxygen, makes the air all the more deadly, and so interferes with and also makes very perilous the work of any rescue party. It is this carbon monoxide gas which has been proved beyond doubt to be the poisonous gas formed in underground fires, and by the firing of explosives, etc.

[1] Baumann: *Report of Prussian Fire-damp Commission*, London. 1893, p. 44.

[2] Thomas: *Coal, Mine-Gases and Ventilation*, 1878.

[3] Meyer: *Jour. für Prakische Chemie*, Vol. IV., p. 42; Vol. V., pp. 144 and 407; Vol. VI., p. 384.

Properties of Carbon Monoxide.

The specific gravity of carbon monoxide is 0·969 or nearly the same as air. It is colourless, odourless, and tasteless. It does not support combustion. It burns with a bluish flame, which may be observed on the top of coke fires, at furnaces, etc. When it burns, carbon monoxide combines with the oxygen of the air to form CO_2. At high temperatures this gas is a powerful reducing agent, uniting with another molecule of oxygen to form CO_2; and advantage is taken of this important fact in many metallurgical processes for reducing the oxides of metals to the metallic state. Unlike the other gases which have been considered, CO is not given off from coal strata except under unusual circumstances, but is commonly the result of incomplete combustion. Poleck, however, using the spectroscopic test, found CO in the ordinary air of a mine under average normal conditions; in one analysis, indeed, it amounted to 1·87 per cent. In the same sample there was 41·49 per cent. of CO_2 which he regarded as produced by decomposition. In our opinion, the percentage amounts of these gases found in these analyses rather indicate some underground fire caused by spontaneous combustion. Meyer, writing on the gases enclosed in coal, found in a German coal which had been weathered, no less than 1·82 per cent. of CO in the gases given off after the coal had been exposed one week in the air. One of the most important points to remember of carbon monoxide is, that it is an explosive agent, and that even traces of this gas (as Lewes has demonstrated) mixed with certain proportions of air, more especially if coal-dust is present, will explode at a lower temperature than a mixture of marsh gas and air.

Deficiency of Oxygen in Mines.

Before dealing with the physiological action of carbon monoxide, it will be well to discuss briefly the subject of *deficiency in the supply of oxygen in mines*, and the effects of breathing rarefied air deficient in normal amounts of oxygen, as for example, on those living at high altitudes, on balloonists, etc.

As a general proposition, it may be said that the symptoms produced by a diminution in the quantity of oxygen and those produced by CO poisoning are not very dissimilar.

It has been already mentioned, when dealing with the different gases found in mines, that in some cases the severity of their action was increased by the lower proportion of oxygen present; indeed in some cases it is this deficiency alone in the amount of oxygen present, and not the presence of the foreign gas, which is the lethal factor.

From what has been said regarding the formation of the other gases

present in mines, it readily follows that mine air is much poorer in oxygen than atmospheric air; indeed, in the return air-course it will be found in some cases to be several units per cent. less. Although this decrease in the amount of oxygen in the return air-ways is partly caused by the consumption of the air by men, ponies, lamps, etc., it is probable that by far the greatest loss in percentage is caused by the oxidation of coal, just as it has been shown that the greatest percentage of CO_2 is produced, not by respiration of men, etc., but by oxidation of oxidisable matter in the mine, and principally coal.

The effect of breathing air deficient in oxygen in mines is just the same as breathing rarified air at high altitudes. Alteration in the density of the air we breathe affects the tension of the oxygen in the blood, and so affects also our respiratory activity. The pressure of the oxygen in the air must be sufficiently great if the hæmoglobin is to take up enough oxygen in the lungs, consequently absorption of oxygen by the blood takes place more rapidly in denser than in rarified air. But it has been found that the pressure can be reduced one half without causing serious symptoms, and also that, if the pressure is increased, the oxygen percentage may be lessened without ill-effects. Consequently in deep mines where the pressure is increased, a smaller percentage of oxygen may cause no inconvenience. When the atmosphere is deficient in oxygen the miners speak of it as "dead air."

As the immediate symptoms caused by breathing air deficient in oxygen are not unlike those produced by CO, we shall describe them in detail. These symptoms are all grouped under what may be termed "*mountain sickness.*" The descriptions of this which have been furnished by various travellers, explorers, mountaineers, and aeronauts, furnish a very good picture of what occurs in man when very high altitudes are attained; that is, where change from normal to low pressure has been comparatively rapid.

It will be seen, in discussing the establishment of toleration to small doses of CO, that although symptoms of slight poisoning are produced at first, yet very soon the organism in a manner protects itself, and the effects of the CO are no longer felt. In the same way, in mines in which the air is very deficient in oxygen, toleration may also be to some extent established. The same is found in breathing the rarefied air of mountains. The rapid transition from a normal atmosphere near sea-level to one much deficient in oxygen (where, for example, high heights are scaled without previous preparation) quickly brings on urgent symptoms, while in persons accustomed to breathing such rarefied atmospheres practically no inconvenience is felt. Conway, in his first essays in mountaineering, felt the effects of the rarefied air when at nearly half the height he could reach with impunity after he had been accustomed to climbing; and to show how far constant training produces toleration of

extremely rarefied air, experienced mountain climbers may ascend 15,000 feet and be only slightly affected. The great traveller, Güssfeldt, when climbing Aconcagua in Chile, which is over 20,000 feet high, only began to feel slight breathlessness when he had attained an altitude of 18,500 feet; that is to say, at a pressure considerably less than half the atmospheric pressure near sea-level. Symptoms of the want of oxygen appear very much more quickly where muscular exertion is being made than when at rest, because during exertion there is a greater demand by the organism for oxygen. In climbing the Andes, for example, where the ascent is made on mules, symptoms do not commence till a very high altitude is reached; and the same is also experienced by balloonists who can reach immense heights without urgent symptoms appearing. Glaisher, for example, in his balloon reached nearly 18,000 feet without suffering much inconvenience. The same thing is found in mines where there is a deficiency of oxygen; the men who have the hardest work are the first to suffer and are those who suffer most severely.

The first symptoms to develop, as the atmospheric pressure becomes lowered, are increased action of the heart and lungs, since the whole organism now cries out for more oxygen. Palpitation with throbbing of the arteries becomes more marked, and breathlessness increases. Headache, which is often of a very severe character, is early complained of, there is a feeling of weakness in the legs, and the traveller feels compelled to sit down and rest. The powerlessness, as we shall see in dealing with CO, comes on before the mind is benumbed; the individual realises perfectly his helpless condition. The symptoms become worse; he becomes giddy; his head swims; there is nausea, and, often, epigastric pain or distress; and with the loss of power in the limbs, a feeling of lassitude supervenes. As the air becomes more rarefied, cerebral symptoms develop, such as irresistible drowsiness, decreased cerebration, confusion of mind, depression, and the individual does not care what happens to him or realise in what danger he may be. The hands and feet become quite cold. Further decrease in the percentage of oxygen causes injection of the eyes, bleeding from mucous membranes (eyelids, nose, gums, and bronchi), lividity of the face, and the development of unconsciousness. All these phenomena vary greatly in different individuals according to age, constitution, etc., and the amount of muscular energy expended. Humboldt,[1] in his graphic description of his ascent of Mt. Chimborazo, tells of his sufferings as the air became more and more rarefied; how he experienced a feeling of nausea accompanied by giddiness which was far more distressing than the difficulty of breathing; and how blood exuded from the lips and gums, and the eyes became bloodshot. "I was seized" he records, "with such violent pain in the stomach and overpowering giddiness that I sank upon the ground in

[1] In Whymper's *Travels among the Great Andes of the Equator*, 1892.

a state of insensibility." Paul Bert[1] quotes cases in which hæmoptysis, hæmatemesis, and diarrhœa with melæna occurred in cases of mountain sickness in Peru, and these are also found in cases of gas poisoning. Glaisher[2] states that at 18,000 ft. he first felt palpitation of the heart, which gradually became violent with greatly increased heart action and impeded respiration, and then his hands and feet became livid. At 23,000 ft. there was great dyspnœa and much nausea. The greatest height reached by Glaisher was 26,916 ft. At this altitude he could hardly move his arms and legs, his sight began to leave him, and although he could still see his companion yet he was unable to speak; then he lost consciousness.

There is another very striking fact in mountain climbing worthy of note, namely, that persons who consume much alcohol are much more susceptible than others to mountain sickness. This is akin to the fact noticed by us that alcoholics are also very susceptible to poisoning by carbon monoxide.

Paul Bert's experiments on animals bear out the experiences of travellers in heights, that there is a distinct lowering of bodily temperature where the oxygen percentage falls very low. This is a symptom which is also produced by CO poisoning, the practical significance of which will be emphasised when discussing treatment.

The Effect of High Temperature in Mines on the Action of Noxious Gases.

In addition to directing attention towards the gaseous and other impurities present in mines, Haldane and Hill have pointed out how important a factor in mine ventilation is air movement, and the keeping of the temperature below a certain point. Cool air and air movement "promote the metabolism and activity of the body, and by stimulating the cutaneous nerves keep up the tone of the body. The cutaneous sense organs are influenced by the temperature and by the relative humidity of the air which controls the evaporation of moisture from the skin, and this influence has a potent effect on our mental state."[3] Impure gases in the atmosphere of a mine have a much more serious action when the temperature is high, or where there is not sufficient movement of the air to keep it cool. From what has been said, one can realise the importance of having a good movement of air by electric fan or compressed air when dealing with underground fires; and the danger of having men working too long at a time while putting in the dams in such circumstances.

[1] Paul Bert: *La Pression Barométrique*, 1878.
[2] Glaisher: *Travels in the Air*, 1871.
[3] L. Hill: "Rescue Work in Mines," *Colliery Guardian*, 17th March, 1911, p. 527.

Natural Gas.

Natural gas may be found in many places. Carnegie, in 1885, used it in his famous steel works in Pittsburg, deriving his supply from gas wells over 100 miles distant. To-day many iron and steel works use it for fuel whenever they can get it. It was estimated that in America, in 1905, one million householders used it for fuel and light, and over one hundred iron and steel works used it exclusively as fuel. It abounds in Canada in the town and neighbourhood of Medicine Hat, where it is similarly utilised for domestic and manufacturing purposes.

At Heathfield, in Sussex, natural gas was discovered by the Railway Company when searching for an underground water supply, and it is used for lighting the railway station. The consumption is about 1000 cub. feet per day. The gas, owing to its richness in ethane, which means high illuminating power, gives a light of 12 candle power. It was analysed by Dr Dixon, and his analysis may be compared with two analyses from other sources.

	Heathfield Gas.	"Blower" in America.	"Blower" in Austria.
Methane . . .	93·4	92·0	88·9
Ethane . . .	3·0	3·0	...
Nitrogen . . .	2·7	2·0	10·8
CO	...	...	...
CO_2 . . .	...	...	0·3
Hydrogen . .	...	3·0	...

Unpleasant accidents sometimes arise owing to its presence. At Bo'ness, in 1911, the residenters in a district made complaints about a very strong smell of gas. In order to remedy this, the Gas Company laid down a new main, but there was no improvement. They re-opened a part of the ground, and while doing so, the workmen were alarmed by the whole tract igniting. It was then discovered that the flames resulted from the ignition of gas escaping from the minerals underneath. This explained a mysterious outbreak of fire which had taken place in that district—the natural gas finding its way into the dwelling-house from the ground.

At the post-office in a Staffordshire mining village, a series of violent explosions led to the discovery of natural gas. The postmaster, accompanied by a woman carrying a light, entered a cellar under the house, when a violent explosion took place, the woman being badly burned. Careful examination showed that this gas was finding its way up through the crevices in the ground under the shop floor. The occupier had the floor of the cellar cemented, after constructing a tunnel for the gas to flow into a reservoir which now supplies the house with light and fuel.

CHAPTER III.

Underground Fires.

One can imagine no more serious accident, no more terrifying condition, than an underground fire. From the method of ventilating a colliery and the combustible nature of coal, one can understand that an underground fire may spread very rapidly, and that what only points to suspicious symptoms of a fire (increase of temperature, sweating of coal, etc.) may in a few hours develop into a blazing reality. The gravity of the situation will greatly depend upon the position of the fire in the workings, the most dangerous position in an underground fire being naturally a place between the fire and the exit of the air-current. Where there is only one intake air-shaft the danger is greatest, where there are several the danger to the men is greatly lessened.

Taking the average speed of the main air-current as 400 lineal feet per minute, if a fire were to break out at the pit bottom and the length of the main air-passage were, say 8000 feet, it would take twenty minutes for the gases to pass through the main air-way. This gives one some conception of the serious nature of an underground fire, and of the rapidity with which the poisonous gases may pass.

Underground fires may be the starting-point of serious explosions, large volumes of inflammable gases being generated by the fire, and these, owing to the breaking down of the stopping, or to a crush in an area full of gas, find their way into the roads and become ignited. One or two authorities hold that this is the explanation of the terrible Courrières disaster, although most are agreed that it was a coal-dust explosion. Again, many serious fires, as, for example, in the recent Whitehaven disaster, are started by explosions of fire-damp or coal-dust. An explosion caused by blasting started a fire at Karwin (Silesia), in 1894, and led to the loss of 223 lives.

The extinguishing of a fire in a fiery mine is one of the most difficult and trying problems a mining engineer has to face, and much ingenuity is often displayed by managers in dealing with fires. Not only so, but the fact of these fires being a serious loss to the owners, necessitating the cutting off of large areas of workings, as well as the destruction wrought by the fire, and the possible dangers arising from it, all go to make an underground fire a matter of the greatest possible anxiety to mining officials.

Causes of Fires.—The following are the principal *causes of fires.* These have been taken from the various Reports of H. M. Inspectors of Mines during the past twenty-five years. They fall naturally under two classes, viz. :—(1) Those due to spontaneous combustion, and (2) those due to other causes.

I.—Outbreak of Fire in the Timbering or other Combustible Material in the Pit by Naked Lights, etc.

In the mining disaster on the slopes of Snaefell in the Isle of Man, which resulted in the death of twenty men, and which is memorable for the fact that it furnished the first clear evidence that carbon monoxide gas is the great danger in underground fires, the fire was probably set a-going by a candle carelessly left burning which ignited the dry timber, trees, or supports. When these gave way, the roof collapsed, and consequently interfered with the ventilation of the mine. Many large as well as small fires in pits have undoubtedly arisen in this way. In 1892, in Pibram, Bohemia, a fire broke out which led to the death of 319 men. This also was produced by the ignition of timbering by careless handling of an open lamp. In the same way 101 lives were lost in a pit near Kattowitz (Silesia) ; and in many other districts of the same country these accidents were not uncommon till the general adoption of safety lamps. In Westphalia, forty-six lives were lost owing to the ignition of a rope by an open lamp. We understand that many small fires, which are promptly put out, occur almost daily in pits. These are caused by the open lamps of the miners brushing against dry timber especially in well-ventilated roads, the particles of lighted wick adhering to the timber and causing it to ignite. In the same way, or by the upsetting of a lamp, etc., wooden engine-houses or cabins may be set on fire. Recently a serious fire broke out in the Pancoast mine in Pennsylvania, in the engine-house, which is situated at the head of a slope 750 ft. below the surface. Sixty miners who were working in a blind gangway more than a mile distant were suffocated, as were also two men who were subsequently engaged in rescue operations. The fire had passed from the engine-room along the oil-soaked beams.

At Barfurlong, in 1891, a fire was started in an underground haulage engine-house by an open paraffin torch-lamp setting fire to the floor. It led to the death of sixteen men. At Kinneddar the wooden lining of the pit-shaft took fire. Men were sent down to put in a dam. The flames came back on them, severely burning seven, while two fell down the shaft. The mine in a very short time was a seething mass of flames. At the Cannock Chase coal-field (Dec. 1911) a fire was discovered in the lamp-room, about thirty feet from the shaft. The lamp-house is a wooden structure where a large quantity of oil is stored for the safety lamps It

is supposed that some of this oil had been upset by accident. A number of the coal trucks also caught fire. Five men were unable to escape and were poisoned by the fumes. Open lights carelessly handled near the fodder in stables have also been a frequent cause of underground fires.

A fire broke out, in 1888, in a mine at Cleator Moor. As all means to put it out failed, it was arranged to allow the water up by stopping the pumps. This was done, and next day fifteen men were allowed down an adjoining mine which communicated with the one in which the fire was, with the result that three men were killed, and the other twelve escaped with the greatest difficulty.

In several cases, terrible fires have broken out caused by underground boilers and underground furnaces used for ventilating, which ignited the timber or coal, and in others by sparks from machinery dropping on inflammable material. Fires arising in this way are fortunately not so common nowadays, as power is so much more easily transmitted and mechanical ventilation much more common.

At pit bottoms fires have been frequent, and when they occur there, the consequences may be disastrous, as the fire spreads rapidly owing to the ingoing current of ventilation carrying the poisonous products to all the workings of the mine. At the pit bottom there is generally plenty of combustible material present, such as woodwork, fixings for ropes, cupboards, greasy articles, coal-dust, boards, etc., and as there is usually a good current of air, the conditions are perfect for propagating a fire should one be started by, for example, an upset lamp, A small fire may do a great deal of damage, and it is well to insist on the fact that a smouldering fire may generate large quantities of poisonous gases. No fewer than 139 deaths were caused at Thornhill colliery by a small fire near the bottom of the down-cast shaft. Indeed, any fire, however small, on an intake road is a most dangerous occurrence, for unless means are immediately taken to reverse the air-current, or unless the men themselves escape immediately by the return, the poisonous gases carried throughout the entire pit may lead to their deaths. A number of serious disasters, *e.g.*, the Mauricewood, where sixty-three men perished, have been brought about by the fire breaking out on the main return close to the intakes, and where the two roads were on a steep incline and communicated by leaky ventilation doors. In the Mauricewood disaster the timber at the bottom of a dook in the main return caught fire, with the result that owing to the increase of pressure further up the incline, the poisonous products of combustion were carried into the intake and through the whole mine. It was afterwards found that the men had erected stoppings as they retreated before the fumes, only to find themselves trapped at the end of the return air-way. A peculiar accident which happened at Hartley Colliery, as far back as 1862, conveys a lesson. The mine was ventilated by a furnace, but owing to

part of a pump falling down and wrecking the shaft, the latter was blocked, cutting off the supply of air to the furnace, with the result that the coal, not being completely burned for want of oxygen, evolved large quantities of CO which led to the death of a large number of men. Mention may also be made of the fire at the Hampstead colliery, which occurred in March 1908, and which resulted in the death of twenty-six men, as this was the first occasion in our country in which breathing apparatus, which has lately been invented in such numbers, was put to practical test. The Draeger and the Weg apparatus were the forms then used. A sad feature of the rescue work in this disaster was that one man, in his anxiety to save life, went further than the apparatus could sustain him in safety and perished. Notwithstanding this, it was demonstrated that the oxygen apparatus has a certain sphere of usefulness in rescue work.

Electricity as a cause of fires must not be forgotten, as it is now so largely used in mines for supplying power for coal-cutting machines, etc., hence great care in insulating and casing the wires should be taken. The new rules for the use of electricity in mines should prevent many such accidents. The disastrous fire at Herminegeld shaft (Polnisch-Ostran), in 1896, which led to the loss of sixteen lives, was caused by the breakage of a wire in an electric cable in the shaft; and a fire, by which one man lost his life, broke out in 1910 in Devon Pit (Alloa Coal Coy.), caused by the ignition of an electric pump. Steam pipes are, of course, never hot enough actually to set fire to the timber in contact with them; but the timber may become gradually charred, with the result that the loose mass soon absorbs oxygen (owing to the larger surface exposed) and ignition may result.

II.—Fires Caused by Fire-damp Explosions.

A very common manner in which underground fires are started is by ignition of fire-damp by naked lights or by a defective safety lamp. Accidents in blasting, such as a blown-out shot, or a spark from the igniter in firing the charge, have given rise to many serious explosions and fires. The access of the oxygen of the air to the still uncooled seat of the explosion may also give rise to fires. So long as the air gains access there is the opportunity of repeated explosions, and as long as there are air and combustible material the fire will rage.

III.—Gob-fires. (Spontaneous Combustion of Coal.)

The great majority of fires underground are believed to be caused by spontaneous combustion. The first two classes just discussed have certainly provided more fatalities, but it is probable, as gob-fires appear

to be on the increase owing to deeper workings, etc., that the mortality-rate from this class may increase considerably. Accidents from gob-fires may occur in several ways. For example in Staffordshire, where gob-fires are common, explosions have frequently been met with. The explosion may be caused directly by the ignition of fire-damp from the gob-fire; or, again, the inflammable gases produced by the fire, which has been dammed off, may find their way out owing to the stopping giving way, or by the crushing of the area containing the gases, and become ignited in the roads in various ways. In Dec. 1911, at Bignall Hill Colliery, North Staffordshire, a number of men were engaged in building off a gob-fire which had broken out in the Bullhurst seam, which is considered the most fiery in the district. The fire was detected on a Friday, and on Saturday everything was apparently safe and the work was nearing completion, when suddenly an explosion took place behind the men engaged on the work, and the stoppings which had been erected were blown out. Six men were killed, twelve injured, some of whom were seriously ill, and, in addition, twenty-five ponies perished. The same night a second explosion took place, but fortunately no men were in the pit at the time. All the deaths were due to poisoning by after-damp. The six men who were killed were working some distance from the stopping where the explosion occurred, but were in the direct return current and got the whole of the after-damp through their air-way. A serious explosion occurred in 1912, at Bentley New Colliery near Doncaster, in which one man died, and eight were seriously affected. A gob-fire which had been bricked-up was being dug out and removed under the impression that the fire was extinguished, when it burst into flame and an explosion followed.

At Agecroft, four men who were engaged in shutting off a fire caused by spontaneous combustion in a mine, were killed by suffocation. At a fire in Whitwick in Leicestershire caused by spontaneous combustion in the main intake air-way, thirty-five men were asphyxiated. In 1901, at the Hill of Beath, seven men, who were engaged in shutting off a fire, were suffocated. Examples of this might be multiplied many times, as this is the commonest way in which accidents from gob-fires arise. The name "gob-fire" arose from the frequency with which these fires were met with in places which had been worked out, or in what the miner in England calls "gob," in Scotland "waste."

Causes of Spontaneous Combustion.—The origin of spontaneous combustion of coal or of goaf material is still under discussion; but this we may venture to say, that undoubtedly the chief factor in its production is oxidation of the coal itself. Some hold that the physical properties of coal have more to do with starting oxidation than the chemical properties. As long as a block of coal, exposed to the atmosphere under natural conditions, remains solid nothing will happen. But

if that block of coal be broken into small particles, and be then exposed to the same conditions, heat will be developed spontaneously; that is to say, the physical conditions have been altered, and by altering these, increased facilities for oxidising chemical action have been set up. In this new physical condition the coal offers a much larger surface to the oxidising action of the air. It is well known that coal can take up as much as a hundred times its volume of oxygen. As a result of the oxidation heat is generated, and with this rise of temperature there is an increased capacity for the coal taking up oxygen, and, consequently, oxidation goes on more rapidly till active combustion results. This theory, which is due to Richter,[1] has been proved correct experimentally by Haldane and Meachem,[2] and it is a most important point when we consider that the deeper the workings the greater the frequency of fires, since the temperature increases with the depth.

Coal-dust also tends to accumulate in deep mines, and fire-damp is more commonly found in them, owing to the superincumbent strata preventing its escape to the surface. The amount of air supply has also a most important bearing on the question of spontaneous combustion. If there is just sufficient air to supply the amount required for oxidation purposes, the temperature of the mass will rise, whereas if the volume of air is sufficiently great to carry away the heat as it is liberated by the oxidation processes, then the temperature will not rise but may fall. In other words, the air which is to carry off heat must be greatly in excess of that required to furnish heat.

One essential condition, therefore, in the origin of spontaneous combustion in mines is the presence of small coal. From this, one would expect that fires would be more frequent in very thick, friable, and soft coals, and that in hard coals, such as steam coal and anthracite, fires would be rare. This is the fact. Consequently it is in the "slack" that these fires often start, and some of the most extensive gob-fires have taken place where the coal has not been taken out in great quantity and a large amount of gob left behind, especially where a slight current of air passes through it. Indeed in a very thick, friable, and soft seam, the coal should be left solid or should be entirely worked out.

The older theory that spontaneous combustion was generally started by the oxidation of iron pyrites, and which for a while was discredited, has lately been revived and has gained considerable support. Bisulphide of iron is present in most coal seams. The old idea was that whenever iron pyrites was to be found, gob-fires might be looked for; but this is not so, as coal may be rich in pyrites and yet not be susceptible to fire: in fact, in South Staffordshire district, where fires are very prevalent, the coal contains a very small proportion of pyrites.

[1] Richter: Quoted by Percy in *Metallurgy, etc.*, London, 1875.

[2] Haldane and Meachem: *Trans. Inst. M.E.*, 1898, Vol. XVI., p. 474.

When air and moisture, which are both essential, are present, pyrites becomes oxidised with formation of the ferrous and ferric sulphates; that is to say, owing to exposure to air and moisture, chemical action is set up with the liberation of heat. Haldane and Meachem's experiments with a piece of Bullhurst coal, and several recent investigations respecting the cause of underground fires, more especially in Silesia, prove beyond doubt that the presence of pyrites has something to do with the production of fires. Some authorities hold that the only action that pyrites has is an indirect one, namely that the presence of fine particles of pyrites in a mass of coal when oxidised rapidly tends to break up the whole mass into fine particles, and thus a large mass of carbonaceous material is rapidly exposed to the atmosphere with consequent oxidation and generation of heat. It is also a fact that when pyrites is distributed in a very fine state of division, fires may originate, but that when present in lumps there is little or no danger.

One authority is of opinion that the initial ignition in cases of gob-fires is due to the existence or generation of certain volatile hydrocarbons, evolved by the rapid oxidation of the coal, which take fire at a low temperature. Undoubtedly in a soft coal, and one in which there is a high proportion of volatile matter, fires are frequent, while in hard coals and anthracite, where there is but a small quantity of volatile matter, fires are rare. Some say that the grinding and crushing action of superincumbent weight with its frictional effects produces heat and so accelerates chemical action. Here, of course, as a result of the grinding and crushing there may happen cracks and fissures in the mass of coal with consequent increase in area of oxidation. Others deny that friction from pressure has anything to do with the causation of spontaneous combustion. Faulty methods of working, whereby much small coal is left behind; heavy rock pressure; leaving behind large quantities of timber in old workings; spontaneous heating of packing material; the character of roof (fires are rare with sandstone roof and common with bituminous roof); have all been advanced as additional factors in the production of fires.

Many hold that the working of the deeper seams will give rise to an increase of gob-fires from spontaneous combustion owing to the increase of temperature, dust, and fire-damp. Certainly, most authorities are now agreed, regarding the production of gob-fires by spontaneous combustion, that their origin is not due to a single or individual cause acting alone, but to several complex causes operating together.

Fires in some seams are exceedingly common, some of the worst districts being centred in North and South Staffordshire and Warwickshire. But even in the same seams, fires are much more common in some districts than in others. In the West of Scotland explosions and fires are not common, although the latter are becoming much more frequent in

Lanarkshire, more particularly in the working of the Ell coal which contains a considerable quantity of iron pyrites. In Fifeshire fires are frequently met with, and in the Dysart thick seam the coal on being worked readily takes fire. Indeed in some coal-fields, notwithstanding the greatest care being taken to build-off the affected areas, certain pits have had to be abandoned. An accident, which illustrates very well the manner in which fatalities may occur in connection with gob-fires, took place recently in the Wemyss coal-field which has been much troubled with fires. The fireman in the Lochhead pit went down the Victoria shaft to make the usual examination of the workings, and as he did not return at his usual time, the engineman, becoming anxious, sent two men to search for him. They were met by a strong smell of coal stink, which was noticed coming up the shaft. This pointed to an underground fire. By the time the searchers reached the foot they began to feel the effects of the poisoned air, and they signalled to be raised. The engineman gave the return signal, but apparently they had become helpless, for they could not reach the cage. Relief parties, which set out immediately, found both men dead near the cage, and further on the fireman was found in a resting position with his lamp still burning, which showed conclusively that it was CO which had led to the death of these three men.

An accident occurred recently which points to another danger from fires underground. When a colliery is worked out or abandoned, no attempt is made to put out existent underground fires, with the result that fires in the workings may go on for months or even years with the accumulation of large quantities of poisonous products, chief amongst which is CO. This gas may travel long distances through fissures, and may, indeed, gain entrance to inhabited houses through drains or weak foundations, with disastrous results to the inhabitants. Such a case occurred recently at Cradley Heath, near Birmingham, where eleven people were poisoned. These persons lived in a row of six cottages which had been built as recently as 1897, but on newly-made ground. The gas had made its way from a subterranean fire which was not far from these cottages. Now in cases such as this, it is generally found that it is the occupants of the ground floor of houses who suffer most, as it is only there that the gas accumulates in sufficient quantity to do damage. As the bedrooms in these cottages were upstairs, it was only when the people came downstairs in the morning that they were affected. In the house where something unusual was first discovered, a man coming downstairs in the morning found his wife, son, and daughter lying in an unconscious condition on the floor, and when the alarm was made and the occupants of the other houses were roused, it was found that seven others were also in an unconscious condition.

An interesting and instructive case of poisoning by CO from under-

ground fires is reported from the Staffordshire district by Chief Inspector Atkinson. A miner was engaged in driving a heading through old workings in the thick coal to "hole" into an old shaft. At the time of the accident the heading was within a few yards of this shaft. The heading was forty yards in advance of the other workings and was unventilated for that distance. Two men were at work here, but one was ordered away for some timber, and on his return found the place in darkness, and his own candle also went out. Groping about he found his mate dead. The Inspector examined the place and found it full of black-damp. It was stated that there had been no gob-fire observed in the pit. The Inspector was not satisfied that death was due to black-damp poisoning (as advanced by medical evidence at the inquest), since air which contains a portion of black-damp sufficient to put out a light may be breathed for some time without any poisonous results. A sample of air was sent to Dr Haldane for analysis. The result of his analysis per hundred parts was as follows :—

Air	Oxygen	14·49	69·23
	Nitrogen	54·74	
Black-damp	Nitrogen	27·135	30·225
	CO_2	3·12	
Fire-damp (CH_4)			0·40
Carbon Monoxide (CO)			0·115

Dr Haldane in his report held that CO was the cause of the accident, the small percentage of CO being virulent owing to the diminished amount of oxygen present in the air. This made the man drowsy, caused his legs to give way, and, probably later, the percentage of black-damp and carbon monoxide increased and poisoned him. The Inspector could not account for the presence of the carbon monoxide in the analysis of Dr Haldane; but a fortnight afterwards the mystery was cleared up, as "gob-stink" was found coming out of the head and roof of the heading, necessitating the building up of this part. The reason we give details of this accident is because we consider it a most interesting and instructive one. Most important lessons may be drawn from it; the most important probably being the value of science in throwing light upon an obscure point in mining.

What are the signs which point to the presence of an Underground Fire?—Fire may break out so quickly that it frequently arises before any suspicious sign is detected; this being especially so in the return air-ways. But, generally, premonitory indications are present for some time before the fire actually arises; for example, beads of moisture, so-called "sweating of the coal," may appear on the coal or "waste," and the temperature of that part of the mine becomes increased. A very early sign which is recognised by skilled miners is a peculiar musty smell, which *Hughes*[1] describes as being not unlike the odour given off by a

[1] H. H. Hughes: *Trans. Inst. M.E.*, 1893, Vol. V., p. 393.

collection of old parchment deeds. If the fire goes further, there is a continuous increase in the temperature of the mine in the neighbourhood of the fire. This becomes oppressively warm, and the men in the neighbourhood may begin to complain of oppression and of slight difficulty in breathing, while their lamps may burn badly. At the same time a peculiar odour is developed, which is at first like singeing, being musty and not unlike paraffin; but later becomes more distinctly tarry and sulphurous in character, the so-called "gob-" or "fire-stink." This is quickly succeeded by smoke issuing from the workings. The smell is due to the volatile hydrocarbons produced by the destructive distillation of coal and to sulphur compounds. "Gob-stink" is often manifested several days before the fire actually breaks out; but it is wonderful how quickly after these suspicious signs are noticed that the place actually shows combustion. In several serious fires it has been pointed out that when the roads are long and tortuous and the fire far distant from where men are working or passing, the smoke loses its characteristic odour and, perhaps, may be quite odourless. For example, in the Snaefell disaster the poisonous gas was upon the men before they realised their danger, only one or two being able to detect something unusual, these declaring that they perceived a very slight smell like singeing.

What is the result of an underground fire, and what is the poisonous gas produced?—When a fire occurs in a coal seam, there is destructive distillation of the coal with formation of hydrogen, carbonic acid, methane, ethane, and other gases of the hydrocarbon group of the paraffin series; and in addition, the production of CO. It is this last-named gas which makes a fire in a pit so dangerous.

At Usworth, a stopping was made in both the intake and the return to cut off the air from a fire which had broken out. A pipe was run through this stopping in the return, and a quantity of the gas therefrom collected by Atkinson,[1] which was tested by Bedson and gave the following analysis:—

CO_2 . . .	4·54	Vols.
CH_4 . . .	8·68	,,
O. . . .	7·23	,,
N. . . .	76·80	,,
CO . . .	2·48	,,
	99·7	Vols.

It is interesting to note in connection with this production of CO that Haldane and Meachem in their experiments found that, in small amount, this gas may be formed from coal at ordinary temperature and without heating, and that at the ordinary temperature coal exposed to air absorbs oxygen and gives off CO_2, CH_4 and a minute quantity of CO.

[1] Atkinson: *Explosions*, p. 113.

The following analyses from Lamprecht,[1] will serve as typical examples of the gases in question. The gases were taken from the draw-off flue at the seat of the fire in the Jacob shaft after complete isolation had been effected.

	CO_2.	Oxygen.	CO.
18th May 1881 . . .	7·33%	7·90%	1·96%
19th August 1881 . .	7·69%	3·69%	0·50%
18th October 1881 . .	6·67%	1·54%	Traces.

Two samples of the air in the Snaefell Mine at the time of the disastrous underground fire were analysed by Dr Haldane. These were as follows :—

	No. I.	No. II.
Oxygen . .	15·48%	15·52%
CO_2	4·22%	4·26%
CO . . .	1·07%	1·10%
Hydrogen . .	0·48%	79·12% (Hydrogen and Nitrogen together)
Nitrogen . .	78·75%	

To show how terribly poisonous the fumes from an underground fire may be, and how quickly they act, Haldane added that inhalation of the above samples would cause loss of power in seven or eight minutes and death in a very short time.

Methods of dealing with Gob-fires.—Many of the accidents caused by gas poisoning at underground fires are caused by the attempts made to extinguish the flames. It will be instructive, therefore, to consider very briefly the principal methods used by mine managers in dealing with this great danger. Regarding these methods, we should add that each case has to be judged by its own circumstances and treated accordingly. The chief methods, however, are (1) Digging-out; and (2) Building-off.

I.—Digging-out.

This is one method which is frequently used, and is undoubtedly the best when the fire is small and easily got at. It is often used in old workings which are easily accessible. Headings are driven into the fire area, the flames are extinguished and the surrounding strata cooled by water under considerable pressure, and then the hot part is dug out and sent out of the mine. In using water to extinguish a fire, it is as well to remember that in this way water-gas may be formed, which, as we shall see, contains a very large percentage of CO. The place is carefully watched for a few days till it cools down. It may then be filled up with sand, etc. This method is certainly very thorough, and, where it can be adopted, is by far the best, for the area so treated will give no further trouble, It cannot be adopted however, where the roof is bad,

[1] Lamprecht: *Recovery Work after Pit Fires*, p. 114.

and it is dangerous in fiery mines ; indeed, great care must always be taken so to arrange the ventilation that no fire-damp is in the vicinity of the dangerous zone. The area, also, may be too extensive to tackle in this way. One can understand how dangerous and difficult dealing with a fire in this way must often be, and in places where at first the method looked as if it might prove successful, it has had to be abandoned for the next method.

Where fires are very common, as, for example, in the Staffordshire district, preventive measures are taken. The air is excluded from the goaf by constructing clay walls right across the waste and across the packs at regular distances. Water is laid down with a small hand engine to immediately extinguish any fire, and stoppings are erected which would enable partial changes to be made at once in the direction of the air-currents. Indeed when a fire breaks out, the whole scheme of ventilation may have to be, and where the fire has obtained a hold must always be, modified.

II.—Building-off.

The method which has generally to be adopted in dealing with underground fires is "building-off," "sealing-off," or "stopping-off" the fire. It is perhaps the safest way, as it is sometimes difficult to forecast what is to happen with a fire; besides, it is scientifically sound. The district in which the fire exists is built-off in order to confine the products of combustion, to isolate, and, in time, to extinguish the fire by oxygen starvation and the presence of CO_2. These stoppings are made sometimes of dirt, but more usually of brick and mortar, are lined with clay, and supported by dirt packings. These are built into the roads leading to the seat of the fire. Several may be built a few yards apart; in general they are built in twos and threes, the space between being filled with sand. The stoppings should be air-tight, and sufficiently substantial and strong to withstand the force of an explosion which not infrequently follows. This method is very successful where the fire area is small, as there is very little chance of explosions occurring; but the possibilities of failure and of serious explosions increase with the extent of the area to be shut off. If this is large, the supply of oxygen will remain abundant for sufficient time to allow of explosive gases being generated by the fire and given off from the strata. As currents of air are set up in the area, these explosive mixtures of gas and air will soon be carried to the fire. Explosions generally occur while the fire is being sealed off, just after the dams have been put in.

If no explosion occurs after the first few days, the danger may be regarded as past, for after that time the supply of oxygen will gradually become less. In 1908, an accident occurred at Glencoe Colliery,

Natal,[1] which shows very clearly the most serious dangers which arise in dealing with gob-fires. The first explosion of fire-damp was a small one, but it ignited a strong blower of gas, which in turn set fire to the brattice, timber, and coal. As a result of a fall of stone, a very large cavity filled with gas was formed, and owing to the outflow of the gas, several small explosions took place at short intervals, the intervals corresponding to the time taken for sufficient oxygen to collect. After a much longer interval, when gas appeared to have accumulated in large amount in the whole area, another explosion of a very violent character occurred, which destroyed several brick stoppings and killed five Europeans and fifty natives, who were attempting to build-off the fire. Thirteen hours after this, another violent explosion took place which killed all the members of a rescue party, including the Inspector of Mines. This explosion started a coal-dust explosion which searched the whole mine.

The building-off of a fire in the return air-way is a very difficult matter. The men should work in relays, and only for a short spell at a time, the length of time depending upon the condition of the atmosphere, when they should be relieved. It is only recently, since Haldane and Hill called attention to the necessity of keeping the air in mines cool and in motion, more especially where gaseous impurities were present, that the importance of this point has been realised in fighting an underground fire. "The closest attention must be paid to the wet-bulb temperature in tackling a mine fire. If the wet-bulb temperature is 99°F. there is no means left for the body to lose heat except by becoming febrile. If the body is 2° to 3°F. above the external wet-bulb temperature, some evaporation of sweat again becomes possible. It takes some time for the mass of the body to be warmed up, so relays of men may work safely for short periods" (Hill). A number of the men who are affected while damming-off a fire may not be overcome by the fumes but by the heat, which is often intense, and they collapse owing to the marked fall in blood pressure. If the atmosphere is very moist, as it will be when water has been used to extinguish the flames and cool the area, the men will be overcome very quickly by the heat if they are allowed to work for too long a spell at one time. Hill says that the rapidity of the pulse is the best guide in such cases. "A pulse which is rapid (say 130), and remains rapid while resting, indicates that caution is demanded." Fresh air should be carried to the men by means of air-pipes. Where compressed air is used for the coal-cutting machines it will be found very valuable in keeping the place cool, and advantage may be taken of electricity to drive a fan in the place. It will be found that these measures will help the men considerably,

[1] W. T. Heslop: *A Natal Colliery Explosion and Underground Fires in Fiery Mines.* Trans. Inst. M. E., 1909, Vol. XXXVIII., p. 338.

and they will be able to work for longer spells at a time. Failure to observe such precautions has often led to fatal results. An accident such as the following should never happen. At Dudley colliery a fire broke out, and the twelve men who comprised the night-shift made efforts to dam-off the place, but they were driven off by the fumes. *On reaching the surface* it was found that two men were missing. A rescue party found the two men dead.

Even when every care is being taken, the men who are "building-off" these places run grave risks of being poisoned by CO, and every now and then we read of a fatal case. We have seen several cases of poisoning in men dealing with such fires. One of these occurred in a town in Lanarkshire where men were engaged "building-off" a gob-fire. Two of these lost their lives, and several others were so seriously affected that it was months before they recovered; two, indeed, have been permanently affected. As these cases are most interesting and unusual from a clinical point of view, we shall deal with them fully when treating of the symptoms of CO poisoning in mines.

CHAPTER IV.

Explosions in Coal Mines.

Since Sir Humphrey Davy and Dr Clanny introduced safety lamps nearly a hundred years ago, many experiments have been carried out and much scientific work done to find out how explosions in collieries arise, and how they may be prevented. Although a considerable proportion of such accidents must, for several reasons, be inevitable, many, on the other hand, might be prevented. The great difficulty in getting the miner interested in technical education, ambulance work, etc., and the want of co-operation between managers and men which result in regulations and rules being ignored or broken, all tend to produce this deplorable state of affairs. It is to be hoped that with wider knowledge of the use of explosives and their risks, of the great dangers of coal-dust, together with more thorough knowledge of the elimination of these menaces, precautions may be taken which will lead to a great reduction in the loss of life from explosions.

Explosions arising from fire-damp and coal-dust caused 21 per cent. of the deaths in mines between 1851-1894 (forty-three years). But the mortality varies greatly; for example, in 1866 there were 651 deaths, while in 1888 there were only forty-nine. In many cases this difference is easily explained, since a severe explosion may cause in any one year a very large number of deaths. The average annual number of deaths from this cause is 220 (Atkinson).

Table III.

Period.	Average No. of Fatal Explosions per Annum.	Average No. of Lives Lost per Annum.
1851-60	82·0	244·1
1861-70	65·5	226·7
1871-80	42·5	268·6
1881-90	24·5	166·1
1891-94	18·0	165·2

This Table is taken from a presidential address by Mr Atkinson to the Mining Institute.

TABLE IV

Death-rates from Explosions of Fire-damp or Coal-dust per 1000 workers employed.

Years.	Explosions.	Falls of Roof, etc.
1851-1855	1·280	2·016
1856-1860	1·234	1·846
1861-1865	·618	1·714
1866-1870	1·158	1·578
1871-1875	·516	1·210
1876-1880	·811	1·132
1881-1885	·408	1·108
1886-1890	·312	1·015
1891-1895	·244	·798
1896-1900	·108	·785
1901-1905	·117	·754
1906	·076	·773
1907	·057	·775

Note.—The figures for 1851-1860 are for coal mines only; for 1861-1872 are for coal and ironstone mines; and from 1873 onwards are for all mines under the Coal Mines and Metalliferous Mines Regulation Acts.[1]

Deaths from explosions, etc., compared with deaths from falls of roof, show slower improvement, and in later years have also become uniform or stationary.

TABLE V.

Death-rate per 1000 Workers due to Explosions.

Year.	Rate per 1000.	Year.	Rate per 1000.
1896	·31	1902	·092
1897	·033	1903	·020
1891	·046	1904	·031
1899	·089	1905	·251 (National Dis.)
1900	·070	1906	·076
1901	·188	1907	·057

In 1896, the first "Explosives in Coal Mines Order" was in operation with its introduction of permitted explosives. For example, safer explosives were substituted for blasting powder in all dangerous mines, the result of this being visible in the figures given above. Besides, since the influence of coal-dust in originating and propagating explosions was realised, the decrease has been considerable. Undoubtedly, also, the science of ventilation is much better understood, and mines are better ventilated. Although there has been a marked decrease in the

[1] *Royal Commission on Mines, Second Report*, p. 84.

number of smaller explosions, it has been shown that, prior to 1860, 2·98 lives were lost in each explosion, whereas up to 1898 the rate had increased to 6·25 lives per explosion.

Against a certain amount of improvement and knowledge must be put, more especially for the future, the necessity of working the lower seams, and consequently having deeper mines, with all their added accompanying dangers. The roadways are also now very much longer and more intricate, and owing to increase of traffic there is much more coal-dust, the result being that the force of the blast will probably be intensified, more damage will be done to the roof, and the resulting falls will greatly interfere with ventilation—all of which, besides, will tend to make recovery-work all the more dangerous. The following table will show the number of deaths from explosions in mines under the Coal Mines Regulation Acts for the twelve years (1896-1907), compared with deaths from other causes of accident.

TABLE VI.

Cause of Accident.	No. of Separate Accidents.	Deaths.	Percentage of Total No. of Deaths.
Explosions of Fire-damp and Coal-dust . . .	218	819	6·5
Falls of Roof and Sides .	5725	5918	47·0
Shaft Accidents . . .	772	893	7·0
Accidents with Explosives .	298	308	2·4
Haulage Accidents . .	2270	2305	18·2
Other Accidents Underground	684	802	6·4
Surface Accidents (including Sidings)	1521	1544	12·5
Total . .	11,488	12,589	100%

When we come to consider the terribly disastrous effects which an explosion in a coal-mine produces, we must bear in mind the peculiar conditions which exist underground, and the manner in which collieries are ventilated. Any explosive effect must be magnified many times by the fact that, when it occurs, it is in tunnelled roads where the air, at the time of the explosion, must in a manner be confined. We know that if gunpowder is sprinkled over a road and fired, it has practically no explosive effect, but it is quite different when it is limited to a small space and confined. We know, further, that as the roads widen, the force of the explosion tends to spend itself, and that the explosive effects are not seen so often at the actual face where the air area is extensive. In coal-dust explosions there is another danger in the very efficient artificial

ventilation which is now found in so many pits, the large volume of air and velocity of the current sometimes being a source of danger rather than of safety. The high velocity of the flame may carry it over many yards of road where there is comparatively little dust. This has been proved by the abundance of the coked dust on the timber in such roads. The high velocity also carries the poisonous air with great rapidity to the unsuspecting men working perhaps a mile or more from the explosion. It was held at the Carolinenglück colliery explosion that it was impossible for all the workings and galleries to have been filled with sufficient coal-dust to carry the explosion four or five thousand yards, and the extension of the explosion was therefore attributed to the artificial ventilation which carried the air, laden with a high percentage of CO, with great velocity to men working nearly two miles away and suffocating many of them.

TABLE VII.

Principal Colliery Explosions in Britain since the Blantyre Explosion in 1877.

Name of Mine.	Situation.	Date.	No. Killed.
Blantyre	Lanarkshire	1877	207
Haywood Wood Pit	Lancashire	1878	189
Abercarne	Monmouth	1878	268
Riscas	Do.	1880	120
Seaham	Durham.	1880	164
Naval Steam Coal, Penygraig	Glamorgan	1880	101
Clifton Hall, Trencherbone Steam	Lancashire	1885	178
Mardy	Glamorgan	1885	81
Udston	Lanarkshire	1887	73
Llanerch	Monmouth	1890	176
Morfa	Glamorgan	1890	87
North Navigation Park Slip	Do.	1892	112
Combs Pit (Thornhill)	Yorkshire	1893	139
Albion	Glamorgan	1894	290
Universal	Do.	1901	81
National	Do.	1905	119
Maypole, Wigan	Lancashire	1908	70
West Stanley	Durham	1909	167
Whitehaven	Cumberland	1910	136
Hulton	Lancashire	1911	344
Cadeby	Yorkshire	1912	87
Universal	S. Wales	1913	429

Causes of Explosions.

Having already considered in detail several conditions which lead to explosions, more particularly the ignition of fire-damp and, also, the gases

which arise from an underground fire, it is necessary here to point to an important factor in the causation and propagation of colliery explosions, the importance of which is now universally recognised and insisted upon, viz., the presence of coal-dust. Before treating of this, however, let us draw incidental attention to the almost unique manner in which an explosion was caused in a pit in Nova Scotia. A disused mine had become filled with gas. This was accidentally fired by a discharge of lightning which struck the iron pulley wheels and passed down the steel rope to the cage, which was standing about twenty feet from the bottom, where there was probably an accumulation of explosive gas.

The following tables throw an interesting light on the subject from the statistical point of view.

TABLE VIII.

Explosions of fire-damp or coal-dust in mines under the Coal Mines Regulation Act during the years 1896-1907. (Report of 1909 Royal Commission.)

Cause.	Fatal Accidents.	Deaths.	Non-Fatal Accidents.	Persons Injured.
1. Naked Lights . .	150	296	1449	2015
2. Safety Lamps (defective or opened) .	20	109	38	100
3. Shot Firing . .	31	285	93	233
4. Miscellaneous. .	17	129	23	81
Total . .	218	819	1603	2429

TABLE IX.

Years 1901-1907. Accidents in England and Scotland compared.

	Fatal Accidents.	Deaths.	Non-Fatal Accidents.	Persons Injured.	No. of Coal Mines.	Output in Tons.
England & Wales	76	199	710	999	2789	237,309,018
Scotland	74	97	739	1016	538	44,689,901

The large proportion of accidents from explosions in Scotland compared with England and Wales, *as seen in the above figures*, is very striking.

Coal-dust and Explosions.—In 1844, while conducting enquiries into the circumstances of the explosion at Haswell colliery, *Faraday and Lyell* found partially coked coal-dust in the roads. They compared this with that of unburned dust, and came to the conclusion that the flames

caused by the fire-damp had acted on the coal-dust, giving rise to enormous volumes of fresh gases which exploded in their turn. Much of our knowledge on this subject, however, is due to the pioneering work of W. Galloway,[1] who set forth his exhaustive researches on the influence of coal-dust in colliery explosions. In 1876, he concluded from his experiments that coal-dust is not inflammable in air free from marsh gas, but that a very slight proportion—0·89 per cent. of CH_4—was quite sufficient to cause explosions. Further experiment, however, made him change his views, as he found that many kinds of coal-dust in the absence of fire-damp may be ignited. Then in 1878 came the work of Hall and Clark, who experimented with blown-out shots in a dip level, the floor being covered with dust; and in 1881, appeared Sir Frederick Abel's Report on the result of experiments made with samples of dust collected at Seaham Colliery. The work of these observers proved that the marsh gas flame is propagated along the workings by coal-dust, the flame finding its way along the dusty haulage-roads where there may be no trace of CH_4.

The work which has since been carried out proves beyond doubt that coal-dust is the most important element in causing explosions, much more important even than fire-damp. In 1881 a Commission was appointed in France to inquire into this question, and after most careful experimental work of their own, and a careful scrutiny of all the work previously done, the Commission came to the conclusion that coal-dust, even when as much as 2 per cent. or 3 per cent. of gas was present, had not much to do with the causation of explosions. The result of the work of the French Commission was that most mining experts in England and Germany, as well as in France, came to hold the view that coal-dust in the absence of fire-damp had no danger, and that only in very dry mines with a very gassy coal could the dust increase the effects of a fire-damp explosion.

But the work of Galloway was soon to be substantiated; for the *Prussian Commission in 1887*, experimenting under conditions as nearly as possible similar to those in a working mine, arrived at the important conclusion that coal-dust, even when fire-damp was absent, might extend and propagate the flame projected by a blown-out shot and give rise to serious explosions. *The British Royal Commission on Accidents in Mines in their Report in 1886* also held that a blown-out shot in the presence of highly inflammable dust, even in the entire absence of fire-damp, might give rise to serious explosions, and it was definitely proved at this time that when coal-dust undergoes distillation by heat it gives rise to explosive gases. In 1890, H. Hall carried out further

[1] W. Galloway: *Proceedings Royal Society*, March 2nd, 1876, Vol. XXIV., p. 239; *Proceedings Royal Society*, March 13th, 1879, Vol. XXVII., p. 410.

experiments, which proved that blasting with gunpowder in dry and dusty mines may, in the entire absence of fire-damp, cause serious explosions.

The Royal Commission on Explosions from Coal-dust in Mines, in their Report *issued in 1894*, drew attention to the following points:—(1) The danger of explosions in a mine in which gas exists even in small quantities is greatly increased by the presence of coal-dust. (2) Coal-dust alone, and especially fine dust without the presence of any gas at all, may cause a dangerous explosion if ignited by a blown-out shot or other violent inflammatory cause. To produce such a result, however, the conditions must be exceptional, and are only likely to be produced on rare occasions. Garforth constructed an experimental gallery at the Altofts Colliery, and the first of the coal-dust experiments were made therein in 1908, which demonstrated the possibility of producing violent explosions with coal-dust without the presence of fire-damp.

The Report of the Royal Commission on Mines (1909) concludes on this point as follows[1] :—"In the starting of a coal-dust explosion two conditions are necessary: (1) A disturbance sufficient to suspend a cloud of fine dust in the air, and (2) the projection of a flame into this cloud of dust."

The reason we have dwelt at some length on the bearing of coal-dust on explosions is, that it is the imperfect combustion of the coal-dust from which comes the CO gas which causes such a large percentage (75 to 92%) of the deaths after large explosions. We have already shown that CO is formed by the ignition of coal-dust in the complete absence of fire-damp. This gas cannot be produced (unless under exceptional circumstances already described) by the combustion of CH_4, thus:—$CH_4 + 2O_2 = CO_2 + 2H_2O$. The partial oxidation of coal-dust and explosions or fires in badly ventilated mines will, however, produce the toxic gas, thus:—(*a*) $C + O_2 = CO_2$; (*b*) $CO_2 + C = 2CO$.

Other important points to be noted are, that the ignition point of gases is lower when the pressure is increased, that compressed oxygen has a much more powerful oxidising action than when under normal pressure, and that great pressure prevails locally in a blown-out shot. *The important conclusion to which we are forced is, that as coal-dust is in the great majority of cases the agent wherein CO originates whereby deaths are produced, if it were removed from the workings of a colliery or treated in such a way as to prevent its being raised in a cloud, the dangers from the ignition of fire-damp would be almost entirely eliminated.*

What happens at an Explosion?—Opinion is not unanimous regarding the origin or the sequence of the phenomena which occur at an explosion, for it must be remembered that we are dealing with a great variety of conditions and surroundings which may considerably modify

[1] *Report of Royal Commission on Mines*, 1909, p. 85.

the effects. The ignition of fire-damp and the conditions which are most favourable to the production of explosions have been already considered. These are not difficult to follow. But it is different when we come to consider coal-dust explosions where the physical and chemical actions are very much more complex.

Since it was proved that coal-dust had to do with explosions, much work has been done on the gases extracted from various coal-dusts by heat. A consideration of the analyses of these sheds considerable light on the subject now in hand. Experimenters find that coals differ greatly in the nature of their enclosed gases, some dusts being rapidly inflammable owing to these gases, and some more so than others. Again, it has been found that the inflammability of the dust may be greatly increased by its absorption of oxygen. *Bedson*[1] heated coal-dust at 100°C, and found that a given volume of the coal produced from ten to eleven times its volume of gas of the following average compositions :—

	I.	II.
CO_2	0·7	0·85
O_2	9·4	6·95
CO	0·1	Trace
Olefines	0·0	1·10
Marsh Gas	16·8	18·40
Nitrogen	73·0	72·70
	100·00	100·00

The coal, after ceasing to give off gas, was powdered and heated for twenty-four hours at 100°C, when it evolved gases of the following composition :—

CO_2	0·85
O_2	6·95
CO	Trace
Olefines	1·10
Paraffins	17·90
Nitrogen	73·20
	100·00

All coals give off hydrocarbons like marsh gas (the so-called paraffins). Sometimes other members of this group besides CH_4 are met with, and an important point brought out in recent experimental work by Bedson and others, where these are met with, is that twenty-three volumes of air to one of this gas are required to form the most explosive mixture, whereas it takes about ten volumes of air to one of methane to form the most explosive mixture; that is to say, it requires a smaller proportion of such gases

[1] Bedson: Trans. Inst. M.E., Vol. XXIV., p. 27.

(4 per cent.) to make the most explosive mixture as compared with marsh gas and air (9·5 per cent.). Prof. Meyer[1] demonstrated that explosive mixtures of oxygen and ethane ignite at lower temperatures than similar mixtures of marsh gas and oxygen. The Austrian Fire-damp Commission also concluded that the sensitiveness of dust to ignition increases with the proportion of easily inflammable hydrocarbons, especially with the amount of ethane liberated at 100°C, and also with the dryness of the dust, and on the other hand, as has been pointed out, where the pressure is increased, as in a blown-out shot, the temperature of ignition is lowered.

Regarding the production of small percentages of carbon monoxide by destructive distillation of coal, it must be remembered that even traces of this gas mixed with certain proportions of air or oxygen, more especially in the presence of coal-dust, form a highly explosive mixture, which will, as Prof. Lewes[2] has demonstrated, explode when ignited at a lower temperature than a mixture of marsh gas and air.

It has been already mentioned that the physical state of the dust is important: the finer it is the more easily will it be fired. Explosions have occurred in flour mills, *e.g.*, in July 1872, owing to the friction of the millstones igniting a mixture of fine flour and air, the Glasgow Tradeston Mills were completely destroyed. A similar accident took place in 1911 in another flour mill in Glasgow. An explosion in a candy factory in New York, a few years ago, resulted in the loss of twelve lives and the destruction of the building. A large quantity of starch is used to prepare the moulds, and the explosion probably arose from the atmosphere becoming charged with fine starch powder. In the same way, an explosion is said to have arisen in a linoleum factory, fine cork-dust in this case being ignited. In 1911, an explosion took place at Bibby's Oil-cake Mills, in Liverpool, in which thirty-eight were killed and nearly one hundred injured. As the seeds which are crushed in the mills contain no oil, an exceedingly fine powder is formed which became ignited. In the same way explosions have also taken place in sugar refineries, storage works, and factories for the preparation of madder, lycopodium powder, flowers of sulphur and other products. In a White Paper issued by the Home Office in March 1913 regarding Dust Explosions, giving the results of experiments with various industrial dusts by Dr Wheeler, chemist to the Explosives in Coal Mines Committee, much valuable information on this subject may be found.

Even coke-dust, which has only a small percentage of gas left, might produce an explosion if the temperature produced by a blown-out shot were sufficiently high to produce CO, which with air and dust would produce a powerful explosive mixture. That very fine dust exists in

[1] Meyer: Quoted in *Prussian Fire-damp Commission's Report.*

[2] Lewes: Trans. Inst. M.E., Vol. IX., p. 320.

mines may be seen in the roofs of most haulage roads where festoons of a fine sooty character are visible; and there always exists in the atmosphere of mines, dust of a very fine, often impalpable character. This dust accumulates to the greatest extent in the main haulage roads owing to the large quantities of coal carried over them, and to the comparatively high velocity of the air and the speed with which the hutches travel by mechanical haulage. The main intake air-ways are generally the main haulage roads, hence the tram roads soon become deeply covered with dust which has to be periodically removed. But the same care is not taken with the dust which has accumulated on the roof and sides. The longer this dust remains the more inflammable it will become. That this accumulation of dust has a great deal to do with the dire effects of explosion is proved by the fact that the damage done is generally greatest in the intake air-ways where there is the greatest quantity of dust. For example, at the Parkslip disaster, in which 112 miners met their death, the flame passed along three hundred yards of the roads. The intake was used as a haulage road and was therefore very dusty, and here it was that the flame passed, while it did not pass along a yard of the return air-way, which was not used as a haulage road, *and was wet.* Instances of this could be multiplied many times. It is to be noted, at the same time, that it is very difficult to ignite dust by the naked flame. In the experimental gallery, it was found that the easiest way to produce explosions of dust was by a blown-out shot of blasting powder, and by allowing the heated products of combustion to come in contact with a dusty atmosphere.

The Phenomena of an Explosion and how Produced.—Many dust explosions have been started by the ignition (in whatever way brought about) of fire-damp and air. This causes a concussion which tends to throw up into the atmosphere of the mine a cloud of fine dust, which in its turn becomes ignited. Or, again, the fine particles of dust from the roof, pavement, and sides are thrown into a sort of whirlwind of closely-packed, dense clouds by the burning gases from a blown-out shot, and these become ignited. The oxygen of the air is used up, and owing to the combustion, an enormous rise of temperature, followed immediately by an enormous expansion, is produced, which is the actual explosion. As a result of this, fresh clouds of dust are stirred up, and the explosion may be propagated over very long distances as long as there is sufficient dust and sufficient air. For example, at the Seaham disaster, in which 164 men and boys and 181 horses and ponies were killed, the explosion traversed the main roads for a distance of 7,500 yards. Indeed in some explosions every part of the mine is searched by the flame. At first it would appear as if the flame travels only with a moderate velocity, with the result that the sooty particles of dust in suspension are exposed for some moments to the flame, and consequently the dust is coked and

gas is evolved. In fact the track of the explosion may be traced by the coked dust on the timbers, etc., which varies from a fraction of an inch in depth to about four inches, as in an explosion in New Zealand. A comparative analysis of the original coal-dust and the coked products will prove that there is a great diminution of the volatile constituents of the dust; just about half the gas, however, being set free.

Prof. Henry Louis[1] has drawn attention to some very interesting work which has been carried out in France by Taffanel[2] in an experimental gallery nearly eight hundred feet long. Taffanel holds that it is necessary for a wide-spread explosion to have the initial or local explosion so violent that the blast of air should travel more rapidly than the flame. If this happen, the dust would be stirred up before the flame reached it, and consequently, ideal conditions would be produced for causing the propagation of an explosion. If the initial explosion were not sufficiently strong, the blast of air would not travel quickly enough, and accordingly the explosion would not be propagated so far.

Since blown-out shots are regarded as a frequent cause of explosions, it is necessary to understand what is meant by the term. In many of the experiments at Altofts experimental gallery a small cannon, loaded with blank shot, was used to stir up and ignite the dust. A blown-out shot has practically the same action as this. A hole is drilled to various depths in the coal, at the bottom of which the charge of explosive is placed, while the rest of the hole is filled up with "tamping" composed of clay or other substance. This is inserted to resist the bursting out of the charge, so that the explosive will expend its energy in disintegrating the coal. Where the "tamping" has not been properly carried out, the coal has a greater resisting power, with the result that the "tamping" will be blown out into the road. There is generally with the blast a certain amount of flame which may lead to the ignition of the cloud of dust which has been raised.

Most authorities hold, then, that there must be a primary explosive force, *i.e.* a small local explosion of fire-damp, a blown-out shot, etc., and that the blast of air should set in atmospheric suspension a thick cloud of dust, what Blackett has very happily called a "pioneering cloud," which in its turn becomes ignited. If the heat were now sufficient to make the pioneering cloud of dust give up its gases, there would then exist an expanding whirlwind of incandescent dust at a high temperature, from which inflammatory gaseous hydrocarbons are quickly being distilled and evolved, which, when present in sufficient quantity and under confinement, explode. Then in succession more clouds of dust are stirred up, and a progressive repetition of the phenomena occurs. On the high

[1] Louis: Trans. Inst. M.E., Vol. XXXIX., p. 747.

[2] Taffanel: *Essais sur les Inflammations de Poussières. Comité Central des Houillères de France.* April and May 1910, p. 73.

percentage of gaseous hydrocarbons, and the character of these produced by the dust, will depend the severity of the explosion.

As has already been noted, traces of CO render mixtures of coal-dust and air highly explosive, and of such a mixture the temperature of explosion is lower than with methane and air. Considerable quantities of CO are, as a matter of fact, produced by underground fires and by explosives, hence it has been advanced by several that *CO is an important factor in the starting of many explosions.* This description of the phenomena of an explosion may be concluded by adding the following quotation from Louis: "Obviously a dust explosion was not to be expected from every shot that was fired. A dust explosion could only occur when a considerable number of conditions were all fulfilled simultaneously. Unless there was finely-divided dust present, unless that dust was stirred up into a cloud with sufficient dust suspended to form an explosive mixture, and simultaneously a source of heat to ignite that mixture was present, an explosion would not occur. It was obvious that the odds against each one of these conditions were considerable, and therefore the odds against all those conditions occurring simultaneously were still greater. When these conditions did occur an explosion of coal-dust would be inevitable." "That is why great dust explosions can only be very rare" (Taffanel).

A good deal of work, which owes its inception to Bunsen, has also been done regarding the phenomena of the explosion of gases, such as the *temperature of combustion*, the *pressure produced*, and the *velocity of explosions.*

Berthelot[1], *and Mallard and Le Chatelier*[2] have done not a little experimental work to show the great velocity of explosion of gaseous mixtures. The former showed that the rate of explosion rapidly increased from the point of origin till it reached a maximum; and Prof. Dixon[3] has carried out a number of experiments which prove the conclusions arrived at by Mallard and Le Chatelier, viz.:—that when certain mixtures, *e.g.* nitric oxide and carbon bisulphide were fired, the flame travelled a certain distance at a uniform velocity, and that, after a certain point had been reached, vibrations were set up which became more and more intense, and either the flame went out altogether or the rest of the gas detonated with extreme velocity. Dr Dixon showed how, when a mixture of coal-gas and air, or fire-damp and air, becomes ignited and passes along a road in a mine, it soon begins to vibrate with more and more intensity till enormous oscillations of heated gas are produced. He held that what takes place in an explosion which starts in the middle of a road is, that the flame would travel slowly for the first fifty or sixty yards before it

[1] Berthelot: *Comptes Rendus de l'Acad. des. Sc.* 1881, Vol. XCIII., p. 20.

[2] Mallard & Le Chatelier: *Comptes Rendus de l'Acad. des. Sc.*, 1880, p. 145.

[3] Dixon: Trans. Inst. M.E., Vol. III., p. 314.

set up vibrations, but at that point the explosion would assume a vibratory character, causing great damage, and that there would be variations in the amount of damage done as the vibrations became spent. This has, moreover, been demonstrated in explosions in collieries; for mutilated bodies are only seen within a small zone of not many yards from the seat of the actual explosion. Little damage is found at the source of ignition, and for a distance of fifty or sixty yards therefrom the bodies of the men do not show injuries by violence, but beyond that point evidences of great violence are likely to be encountered; for example, at 120 yards distance the bodies of men may be found blown into fragments. The explosive blast thereafter gradually expends itself, till there is left just a slight gust of air which could not possibly do any damage, Atkinson[1] holding that lights would not be extinguished three or four hundred yards away.

Dr Dixon[2] has demonstrated the three phases described by Mallard and Le Chatelier of (*a*) ignition, (*b*) vibration, (*c*) sudden pressure or detonation in the following experiment. He took a long vertical glass tube filled with a mixture of nitric oxide and carbon bisulphide. When this was ignited at the open end, the flame passed steadily and quietly downwards to a certain point where the vibrations were set up, which became more and more marked till the rest of the gas exploded violently. Prof. Galloway, in his report on the Whitehaven disaster (p. 41), writes that he once had ocular proof of the fact that the flame does not pass with lightning-like rapidity. It was in a damp mine, and the fire-damp was ignited by a damaged safety lamp. After the explosion the flame passed quickly, but not so quickly that it could not be followed by the eye, along the roof, where at a certain point it stopped. There was a distinct pause, sufficiently long to enable Galloway and his companions to crawl back some twelve or thirteen feet into a larger chamber sixty yards long, to run a short distance sideways from the direct line of the passage, and to turn round and wait for several seconds longer, before a larger blue flame shot out as if through the nozzle of a blow-pipe to a distance of thirty or forty feet or more, with a roar like thunder. Having continued for perhaps ten or fifteen seconds, the flame was then sucked backwards out of sight, followed by a supply of fresh air; then after a similar interval it shot out a second time, was again drawn back to be shot out again, each succeeding time being feebler than the first till it finally disappeared.

We have dwelt at considerable length on the phenomena which occur at an explosion because they throw considerable light on the manner in which injuries to the men are produced.

Damage done above-ground by Explosions.—Sometimes the first intimation to the outside world that a serious explosion has taken place is a terrific roaring noise, and, at the same time, a huge column of soot

[1] Atkinson: *Explosions*, p. 117.

[2] Dixon: Loc. cit., p. 314.

and smoke is seen to shoot suddenly from the shaft, generally the downcast shaft, the clouds of soot falling over the surrounding fields perhaps for a considerable distance. Galloway in his Report (p. 95) on the Penygraig disaster in which 101 out of 106 died, describes the noise as "an ominous report which sounded like a single loud, deep-toned clap of thunder, but it had a weirdness about it that made everyone who heard it tremble." Sometimes there is a sheet of flame, and, in certain cases, dense smoke with its poisonous gases may continue to arise from the shaft for a few hours. The force of the explosion has been known to cause buildings within a few miles to tremble as in an earthquake. Great damage may be done to the pit-head; indeed, it may be completely wrecked. The force of the explosion may carry away the winding gear, dislocate the cage, and send it crashing to the bottom. The fan-house, if not properly protected, is often severely damaged, the roof being blown off and the fan wrecked. Where this happens, the restoration of the ventilation will be seriously interfered with. Usually it is the down-cast shaft which is most affected. The up-cast shaft generally escapes, unless it is used for winding coal and, in consequence, has the necessary amount of coal-dust lying about.

Underground, the damage done may be very great, the main intake roads being generally most damaged, it being along these that many of the bodies are found. At the Seaham explosion, for example, where 104 lives were lost, the whole field of the explosion was confined to the "intakes," very trifling damage being found in the "returns." The latter, unless used for haulage, generally escape untouched. The safest places generally are the return air-ways, which remain for a considerable time free from after-damp. The face in many cases is also clear for a considerable time. But many explosions are on record where the whole mine was searched by the flame of the explosion. In many cases also, the timber props are carried away and the roof falls in, these falls of themselves causing sometimes a certain number of deaths; the pavement rises; rails are torn up, twisted, and bent into various shapes, and sometimes the hutches are found buried deeply in the solid coal; tramways are torn up, rails, sleepers and débris being mixed together as though shot out of a gun; all timber is simply wrecked—for example, the wooden bratticing and the air trap-doors necessary for the guiding of the air-current are shattered to pieces; air-bridges for carrying the air across, and permanent brick stoppings, perhaps many feet thick, are blown down. The result of this, as one can readily understand, is the serious interference with the ventilation of the mine; indeed the air no longer travels through the mine, but passes down the down-cast shaft and proceeds by the easiest and shortest way through the blown-down doors to the up-cast shaft. In this way, therefore, the poisonous gases, which are in a manner locked up in the other parts of the mine, cannot be diluted

and dissipated by fresh air; besides, there is the still further interference with proper ventilation by the numerous falls of the roof.

In certain parts of the mine the noise of the explosion may be so slight as not to awaken alarm among the men; perhaps they may be totally unaware of the occurrence of an explosion, especially if they are working in very remote parts of the mine, far distant from the locality of the explosion. In many explosions it has been noted that the sound was limited to a small part of the mine. But, generally, even the men in the parts far removed from the point of explosion are perturbed by hearing a peculiar noise which they cannot account for, or, again, they may notice an alteration or stoppage of the air-current, while in large explosions the men generally feel, even in the most distant parts, a sudden rush or gust of air. At the explosion in Whitehaven in May, 1910, for example, the first indication that anything was wrong was observed by a roadman engaged in timing the last tubs going out at the time, who noted that "all at once the air rushed out"; that is to say, the air-current was reversed. The air was found to be so thick that the men could hardly see, although their lamps burned as usual. The cloud of dust was accompanied by a buzzing noise. Generally the men are sufficiently alarmed, by whatever means, to cause them to rush out of their places at the face, and in doing so often go from what is in many cases a place of safety right into the poisonous air.

Prevention of Explosions.—In the 1909 Report of the Royal Commission on Accidents in Mines, the means for preventing coal-dust explosions were classified into: (1) those which aim at preventing the initiation of an explosion, and (2) those which prevent its extension. *Primary preventive measures* should be directed towards the prevention of explosions of fire-damp, and the prohibition of blasting under such conditions as may initiate an explosion. The heated products of a blown-out shot have frequently caused explosions. Gunpowder is still largely used for blasting purposes, a special variety, which contains less saltpetre and more charcoal than ordinary powder, being very popular. Any explosive whose temperature of detonation is above 650° C, and which shows flame, is capable of igniting the dust and of distilling the explosive gases. Explosives such as blasting powder should not be permitted underground. We have already seen that coal-dust may propagate a small local explosion of fire-damp, from which may develop a disastrous and far-reaching disaster. *Secondary methods* are directed against the coal-dust. It may be prevented from being deposited, or when deposited it may be treated by watering with calcium chloride solution or with an inert stone-dust, and so be rendered harmless. or it may be removed. It has been found if certain zones are kept clear of dust, that this is often sufficient of itself to stay the advance of an explosion. Where electricity is used, there must be thorough insulation of the electric

current. There must also be perfect discipline in the mine, and loyal co-operation of the officials and men in preventing rules and regulations being disregarded or violated.

Before treating of the causation of deaths in explosions, the action of explosives, so far as they lead to cases of gas poisoning, may be first considered briefly.

Gases Produced by Explosives, and their Action on the Men Breathing them.

Although large numbers of fatal accidents occur annually from the use of explosives, from boring-out miss-fires, thawing nitroglycerine compounds over a fire, etc., we shall consider only those accidents which occur from the inhalation of the poisonous fumes. Minor accidents from such are very common, and most medical practitioners in colliery districts meet with such cases. In most instances, perhaps, these are due to carelessness on the part of the men, who return to their working places too soon after blasting, and when, as they say, their "place was still hot," or "had not cooled down." For example, in 1911, at Bothwell Park colliery, Lanarkshire, three men were engaged in blasting for the purpose of making an air-inlet from a seam 15 ft. above. After the shot had been fired, the man in charge of the place returned to find out the result, when he was overcome. As he did not return, his two mates went into the place, which they found full of dense fumes, and there they found the man insensible. They attempted to drag him into the air-way, but one of them dropped senseless. The remaining man got both of them out; but the one who was the first to be overcome died.

Reports like the following are also far from uncommon. In January of 1911, two miners who were working in an air-way at Hazelrigg eolliery, Northumberland, were overcome by the fumes from blasting, and one died. Again, the miner may be working in a badly ventilated working where a considerable amount of blasting is going on, the result being that the fumes are dissipated very slowly. In driving through a communication to another road, and consequently where there is no through ventilation, these cases of "gassing" are frequently met with. In the Transvaal gold mines the two processes which are the most dangerous are the "raising" and "driving," the "raise" and the "drive" being cul-de-sacs in which proper ventilation is almost impossible. Accidents may be met with in all mines where, owing to the ignorance of the miner, the conditions under which the explosive is used are not properly carried out, as, for example, in the true boring of the holes, the stemming of the charges, and the correct placing of the detonator at the proper depth. The quality of the explosive used is also very important, as these deteriorate much when they are improperly kept.

The too prodigal use of explosives through ignorance of their action is also responsible for some of the cases. We had one case in which two men, who, being quite ignorant of the amount of explosives to be used (they had just started work in the place and had no previous experience of work in pits), fired no fewer than ten shots where two would have sufficed. Besides these, within the same section, which was badly ventilated, and within the space of one hour, other ten shots were fired. Five men were overcome by the fumes, two of them seriously. We were able, nearly one and a half hours after the accident, to detect CO in the blood of these two men—in one by Haldane's method, and in both by the spectroscope. It is important to remember, even when apparently sufficient time has been allowed for the fumes to be dissipated, that when the men do return and begin to work, they may often be overcome by the gases, which have remained in the interstices of the coal or rock, and which flow out quietly whenever the dèbris is disturbed. Many minor cases of "gassing" in collieries occur in this way, and miners affirm that this action greatly depends upon the quality of the coal and the explosive used, some explosives being worse than others in this respect.

An example of the serious effects of the action of the gases produced by blasting is furnished by the Crarae disaster in 1886. As it has some important lessons, we shall give a detailed account of what happened. A monster blast by gunpowder was to take place in the quarry at Crarae, and in order to allow the passengers on the pleasure steamer, *The Lord of the Isles*, to observe the results, it was arranged that at a given signal immediately after the blasting, they would be allowed ashore—to see these results at close quarters. About one hundred and fifty passengers were thereupon landed. Many of these arrived at the quarry about twenty minutes after the blasting, a considerable number panting as if they had hurried. Nearly one hundred onlookers had collected in the quarry gorge, and forty of these were immediately rendered unconscious. In addition, others fell down in a state of giddiness without actually losing consciousness, who, after being taken outside the quarry and resting for a short time, quickly recovered. Of the forty who were seriously affected, six died. The chief points in connection with this incident and which led to the disaster were: (1) absence of wind and heavy state of the atmosphere; (2) that no ill effects were experienced until half-an-hour after the explosion, for a number of the inhabitants of the place were there almost immediately after the explosion, and almost twenty minutes before the visitors arrived, without feeling any ill-effects, so that the gases must have flowed out of the interstices of the broken rock ; and (3) that the influence of the poisonous gases was felt simultaneously in all parts of the quarry. It was found in most of these individual cases that almost at once there was loss of muscular power, followed by the patient dropping insensible

to the ground. In the fatal cases death occurred quietly. Some of those who recovered developed convulsions on regaining consciousness; in others there was delirium, after which the patient became drowsy and slept. No secondary complications developed in any case, but in all there was great prostration, and a long period elapsed before they regained their strength. The powder used was found to yield 3·6 per cent. of CO on explosion. Forty years prior to this accident a similar accident took place in the same locality. After a large blast a number of boys went into the quarries at Furnace, about two miles from Crarae, and twelve were rendered unconscious, though all recovered.

The gases from blasting in mines may travel long distances to a blind end, or to a place where the air is stagnant even far distant from the original place of blasting, and there exercise their toxic effect. The explosive used will in some measure determine the character of the gases produced.

When we consider, however, the enormous amount of explosives employed annually in mines, it is wonderful how relatively few fatal accidents by gassing from the fumes are met with in this country. Until quite recently such accidents were deplorably frequent on the Rand in South Africa, and in Australia. For example, in 1904-1905 there were thirty accidents on the Rand, in which forty men died and twenty-four were seriously affected: and we have been informed by "shift-bosses" that a very large number of minor cases occur which are never reported. There, many of the gold mines are ventilated chiefly by natural means. Perhaps the large use of compressed-air drilling machines has encouraged a certain amount of carelessness in providing proper schemes of ventilation, as the exhaust air is useful in keeping cool the atmosphere at the face. It is a common custom to blow through compressed air to clear the fumes in poorly-ventilated workings. This is undoubtedly most useful where developing operations are going on at the end of "drives" and "rises." The presence of a high percentage of CO_2 and CO in many of these mines shows how serious was the pollution of the air. The Transvaal Phthisis Commission in their Report found, as the conditions in the mines in the Transvaal differed so much from those in home mines owing to the much greater proportions of explosives used, that the amounts of CO_2 alone should on no account be accepted as a guide to the purity of the air; that it was as important, indeed more important, to find how much CO was present. The following is a summary of a large number of analyses of air in different mines carried out for that Commission.

[TABLE X

Table X.

Summary of Analyses.

	Mine Air.			Pure Air.
	Normal.	After Blasting.	Compressor Air.	
	Average of Six Samples.	Average of Seven Samples.	Average of Three Samples.	
Oxygen .	20·38	19·90	20·76	20·96
CO_2 . .	0·11	1·59	0·05	0·36
CO . .	0·13	0·39	0·08	—
Hydrogen .	0·48	0·32	—	—
Nitrogen .	78·88	77·57	79·12	79·0
NO . .	0·0004	0·0078	—	—

Not only, then, was the ventilation bad, but the Mines Inspectors drew attention to the fact that the extensive use of nitroglycerine explosives (they are very prodigal in the use of explosives in South Africa), some of which were of inferior quality, had also to do with this serious state of affairs, more especially if the charge were imperfectly detonated, or where too much was used for the work to be done. In many of the mines the men are on piece-work, which may account partly for the miners returning to their places too soon after blasting.

In development work, explosive charges are very heavy. On an average thirteen holes, each 5 ft. 6 in. in depth, are drilled in the end or face of a drive and charged with blasting gelatine. At each blast the average amount of explosive used is about 50 lbs. Each charge dislodges from 12 to 16 tons of rock from the face of the drive, a considerable amount of which is in a fine state of division. Not infrequently the blasting of the development face is performed in two stages *in the same shift*, the second stage being separated from the first by a very short interval of time. Where this method of working is adopted—and this was the general custom on the Rand until quite recently when the mine captains found their output was in no way affected by adopting the plan of one blast per shift—the miners are exposed to greater risks from the inhalation of dust and fumes; and this is, probably, the chief reason why so many development workers fall victims to miners' phthisis. It is true that the miners are not permitted to return to their working-places until the fumes of the blast have become dispersed, but this often means merely until the irritating fumes have disappeared. But CO gas may remain for a

much longer time locked up in the débris, only to flow out, however, when the débris is disturbed during removal.

We have been informed by South African miners that cases of "gassing" occur sometimes many hours after the blasting, where, for example, the place has stood twelve hours, the fumes which were locked up in the blasted rock become liberated when the men began to move it. From analysis of the air in badly-ventilated sections in the gold mines, it was proved that some of the working places were unfit for men to work in them for many hours (over twenty-four in some cases) after blasting operations. As may be readily understood, therefore, the risk of gassing will become very much increased when double blasting in the same shift is performed.

Irvine and Watt,[1] who have had large experience of miners' phthisis and of gas-poisoning cases, are strongly of opinion that this double blasting per shift "has been a very important factor in the higher incidence of miners' phthisis amongst machine drill, and especially amongst development miners."

Moreover, in the evidence led before the Transvaal Miners' Commission of 1912, it was generally agreed that "double blasting in the same shift should be prohibited altogether."

The following analyses, taken in places shortly after blasting, will enable one to realise the degree of pollution of the air from CO gas.

TABLE XI.

Sample taken	20	minutes after explosion . .	CO. 0·01%
,,	5	,, . .	,, 0·63%
,,	7	,, . .	,, 0·68%
,,	13	,, . .	,, 0·54%
,,	15	,, . .	,, 0·55%
,,	9	,, . .	,, 0·23%
,,	10	,, . .	,, 0·90%
,,	15	,, . .	,, 0·13%

In the first three of these samples Transvaal blasting gelatine was the explosive used, in the others Nahnsen's blasting gelatine.

It had long been held in South Africa that it was the nitrous fumes which caused the deadly effects after blasting operations, just as it was held in our own country that it was the sulphurous fumes in blasting with gunpowder; but the large number of analyses carried out for the Commission shows that the proportion of *nitrous fumes* is very small. "Possibly the undue prominence given to the presence and evil effects of nitrous oxides in the mines is due to the pungent odour and immediate irritating action on the mucous membrane which would tend to mask the existence of the more poisonous but less noticeable CO."

[1] Irvine and Watt: "Miners' Phthisis," *Transvaal Med. Jour.*, Sept. 1912.

Most observers are agreed that exposure to the fumes from blasting is a subsidiary cause of miners' phthisis. Haldane, in his report on the Health of Cornish Miners, although he recognised the possibility of poisoning by carbon monoxide during blasting operations, was not inclined to place much importance on this as a co-operative factor in the production of the phthisis of miners. Summons, on the other hand, in his Report on Miners' Phthisis in Bendigo (1907), held that this was a contributory cause, "although," he adds, "these (fumes) are not sufficient of themselves to bring about lung disease." Both of the Transvaal Commissions on Miners' Phthisis, moreover, arrived at the same conclusion. Watt and Irvine, in their discussion of the point, declare as follows:—"It is, therefore, reasonable to assume that an intermittent exposure to smaller amounts of these fumes, which is practically inevitable where the practice of double blasting on the same shift is employed, will tend to create and maintain catarrhal conditions in the air passages and lungs, and will at once prepare the way for and aggravate the effects produced by the irritation of the inhaled dust."

We shall return to this subject when dealing with pneumonia following the inhalation of the fumes from blasting.

The serious reports of the Inspectors brought about a better state of affairs, and now many of the mines are most efficiently ventilated by mechanical means; only in this way, indeed, can the fumes generated by blasting operations be effectively dissipated Further, the supervision of the mines is now very much better with regard to the use of compressed air, a better class of explosives is used, and greater care is taken in storing and firing them. In all the mines in which artificial ventilation has been adopted, it has been found that, where under the old methods of ventilation some hours were necessary to clear a shaft of noxious gases, half an hour now suffices.

A most remarkable case of poisoning, which was recorded by Dr A. R. Sedoux in the Trans. American Inst. M.E., 1903, drew attention to the fact that the use of low flash-point lubricants in the air-cylinders of compressors was dangerous, and might lead to serious contamination of the air. In this case oil had collected in the pipe from the compressor to the receiver, and also in the latter. The compressor became so heated that this oil was set on fire, with the result that the poisonous products of combustion (CO_2 and CO) were carried by the pipe to the men underground, who in this case were depending on the compressed air supply. This led to the death of two men, four others escaping with the greatest difficulty. In 1899, two miners, while engaged in boring shot-holes in the Concordia iron-ore mine near Dermbach with a compressed-air drill which was working under a pressure of four to five atmospheres, perceived a curious smell and felt ill; one managed to get out in time, the

other died. In this case rape oil had been used to lubricate the air-compressor cylinder. Experiments were thereafter carried out, and it was found that this oil yielded CO when the distillation was carried out under pressure and at a high temperature. Some of the deaths from CO poisoning in the mines on the Rand have also been put down to the men breathing contaminated air supplied in the compressed air which is used for the rock drills as well as for ventilation. The CO in these cases was doubtless produced by the lubricating oil becoming heated in the compressor.

In an Appendix to the Report of the Transvaal Miners Phthisis Commission, a number of analyses of the air supplied by the compressor is given. In two mines, dangerous quantities of CO were found in the compressor air; in one case 0·10% CO and 0·0002% NO and in the other 0·13% of CO. The Commission regarded this contamination as a very serious matter, and they recommended that only oil of very high flash-point should be used with air-compressing cylinders; "that the lubricants should be of such a nature that they are incapable of being decomposed or of giving off injurious products under the conditions they are subjected to in the air cylinder of the compressor; and also that the air in-take be taken from outside the engine-house in order to secure a pure supply" (page 14).

In connection with this subject it should be added that Dr Haldane[1] has carried out a number of experiments with three compressors. Different lubricating oils were used, some of them of the cheapest kind, and the compressor was driven hard so that it became very hot. Analysis of the air, however, showed no trace of CO, and Dr Haldane held that it was very improbable that CO could be formed under ordinary circumstances.

The action of an explosive must be kept in mind. It is a chemical action which is set a-going, by combustion in the case of gunpowder, by detonation as in ammonite, etc., and in another class the explosion is partly due to combustion and partly to detonation, such as in the various forms of gelignite, carbonite, etc. Here there is first detonation of the nitroglycerine, then, owing to the heat and flame formed, combustion of the carbonaceous matter. The result of this is that the explosive, which may be either in solid or in liquid form, is immediately converted into gases. These gases, owing to their occupying an enormously greater volume than the original substance,—a volume, besides, which is still further increased by the heat generated,—have powerful disintegrating properties. These explosives depend for their action upon the instability of their chemical equilibrium. They consist of carbon, oxygen, nitrogen, and hydrogen, the oxygen and nitrogen being so feebly com-

[1] Haldane: *Report to Home Department on the Health of Cornish Miners*, 1904, p. 88.

bined that, when fired by detonation or combustion, the nitrogen readily parts with its oxygen to form new compounds with the hydrogen and carbon.

There are different classes of explosives, viz. :—"high" and "low." The former have a considerable percentage of oxygen, much higher than the "low" explosives; the chemical changes take place very rapidly, much more so comparatively than in the "low"; and they are fired by detonation. All explosives, the so-called "safe" as well as the dangerous, give off more or less noxious fumes; also all contain carbon, and therefore where there is good ventilation and thus plenty of oxygen, CO_2 with a relatively small percentage of CO, is produced, but where there is deficiency of oxygen, and consequently imperfect combustion of the carbon, relatively large quantities of CO are produced. When nitro-glycerine, for example, is properly fired, no CO is produced, but when it or its preparations burn, as much as 30 to 40 per cent. of CO may be formed. According to Berthelot three conditions are necessary for an explosive: (1) rapid chemical changes; (2) formation of a large volume of gas; and (3) a high degree of heat; but in mines any explosive whose temperature of detonation is above 650°F. and which shows flame, is quite capable of igniting dust, and according to the 1909 Report of the Royal Commission, should not, therefore, be used in mines.

Let us briefly consider the composition of a few of the more commonly used explosives, and the gases which are produced when they are fired.

1. **Gunpowder**.—Although gunpowder is not nearly so extensively used for blasting in mines as it formerly was, it is still employed in the form of blasting powder. It is now recognised that it is very dangerous in fiery and dusty mines, since, when it explodes, large volumes of gas

TABLE XII.[1]

Gaseous Products of Combustion from *Low* Explosives (Lewes.)

	Gunpowder in Fine Grain.	Mining Powder.	Blasting Powder (Sarran & Veille).
CO_2	50·62	32·15	49·4
CO	10·47	33·75	20·5
N	33·20	19·03	21·3
H_2S	2·48	7·10	
CH_4	0·19	2·75	
H_2	2·96	5·22	2·0—1·4
O_2	0·08	0·00	
	100·00	100·00	

[1] Lewes: *Mining Explosives.* Trans. Inst. M.E., Vol., IX., p. 320.

at a very high temperature are produced, and solid particles, also at a very high temperature, are projected into the air. If the carbon is only partially oxidised, as in badly-ventilated places, relatively large volumes of CO may be produced; and, in addition, smoke and irritating sulphur fumes are given off. The H_2S is, as we have already seen, a particularly poisonous gas.

Gun Cotton.—While a mine tunnel was being constructed in Flintshire, twelve people were overcome by the gases from a gun-cotton blast.

TABLE XIII.

Gaseous Products of Detonation (*High* Explosives) (Lewes).

	Wet Gun Cotton.	Dry Gun Cotton.
CO_2	32·34	24·24
CO	27·12	40·50
H_2	26·74	20·20
N	14·00	14·86
CH_4	0·00	0·00

The percentage of CO in the detonation of gun-cotton may indeed be larger, even up to 48 per cent. There is now a form of gun-cotton on the market for mining purposes, viz., *Tonite*, in the manufacture of which enough barium nitrate is added to supply sufficient oxygen to completely convert the carbon to CO_2, consequently it is only in very badly ventilated places that much CO can be formed after its use.

Gelignite (Gelatine dynamite) consists of nitro-glycerine, nitro-cotton, wood-meal, and potassium nitrate. It gives off CO_2, 26 per cent.; CO, 7 per cent.; N, 67 per cent. (Orsman[1]). Some authorities hold that even less CO than this is produced. This explosive is very largely used for blasting in mines. If it does not explode but burns only, large quantities of CO and nitrous fumes may be produced (CO = 35·9 per cent., NO = 48·2 per cent.: Haldane[2]). With regard to the use of gelignite in mines, poisoning cases sometimes occur through the miners placing the explosive in contact with their bodies in order to soften it, instead of using a warming-pan, and also by moulding it with their hands until the desired consistency has been obtained.

Carbonite is another explosive in use, and yields CO_2, 19 per cent.; CO, 15 per cent.; CH_4 and H_2, 26 per cent.; N, 40 per cent (Orsman).

Roburite has been very largely used in mines. Formerly it was re-

[1] Orsman: *Notes on the Products and Temperature of Detonation of some High Explosives.* Trans. Inst. M.E., Vol. III., p. 94.

[2] Haldane (Haldane, Martin and Thomas): *Report to the Home Department on the Health of the Cornish Miners*, 1904, p. 89.

garded as a "safe" explosive, but the large number of poisoning cases which have occurred from time to time shows that the same care is as necessary when using it as when using gelignite. So many complaints arose regarding the effects after its use, that colliery owners in Lancashire and in Northumberland appointed Committees to inquire into the matter, and as to how the gassing cases which followed its use might be prevented. The Durham Miners' Association also inquired into the nature and action of the fumes from gunpowder, tonite, and roburite. The symptoms complained of were those which are mainly caused by CO, viz. :—headache, nausea and vomiting, cardiac palpitation and tachycardia, and oppression about the chest with breathlessness. The patients gradually lost flesh, and complained of cold, the face becoming pale, but the lips often bluish coloured. There was loss of power in the arms and legs, the patient's grasp became much weaker, and he staggered when he walked. In all cases the condition was accentuated by fatigue. In some cases, also, there were disturbances of vision. It was thought that some of these symptoms were caused by nitro-benzene, because the fumes had a peculiar bitter-almond smell (which is quite characteristic of roburite fumes). In some of the cases, however, jaundice was a symptom, and this is certainly not common after CO poisoning, although it has occasionally been noted. The following is an analysis of the fumes from roburite :—CO_2, 32 per cent.; N, 68 per cent.; CO, 0·0 per cent. (Orsman). Theoretically, no CO should be produced. In Orsman's experiments, however, there was no carbonaceous matter surrounding the heated gases, therefore there could be no CO; but Dixon has pointed out that when roburite is exploded in coal, a certain amount of CO is produced by the action of the heated CO_2 on the coal.

Drs. Drummond and Hume with Prof. Bedson carried out a number of experiments with the fumes of roburite, and as to the action of these on men. Bedson made a number of analyses of the air from places after blasting with roburite, the gases being collected by means of aspirators. He found that the average quantity of CO in these samples was small, and was so quickly dissipated that, at the end of five minutes after blasting, traces only could be found. He detected a distinct odour of nitro-benzene in some cases, however, but was never able to detect its presence by chemical analysis. He held that the CO found in his analyses was produced by the burning of the fuse, and by the action of the heated CO_2 passing over the coal.

Regarding the use of roburite, the Committees came to the common-sense conclusion that longer time ought to be allowed to pass before the men returned to their places after blasting, and also that there should be good ventilation. If these conditions were followed, roburite was no more dangerous than the other explosives.

Table XIV.

Analyses of Gases Produced by Explosion of Roburite.		
	I.	II.
N & CH_4	79·44	79·57
Oxygen	19·34	20·28
CO_2	0·98	0·114
CO	0·24	0·014

According to Bedson, *tonite*, which is also largely used in mines, gives off by explosion more CO and CO_2 than roburite, and we have been informed by miners that the fumes from it take longer to disappear, as they lurk in the interstices of the rock and coal débris and flow out slowly when the men begin to disturb the coal, thus causing "gassing."

It has again and again been pointed out that one great danger from the use of the higher explosives in mines is the fact that the atmosphere after firing the shot quickly clears, whereas with mining powder there is more smoke, and the miner is forced to wait longer before he can enter his place.

In choosing an explosive for blasting in mines, those which produce poisonous gases in large quantities, such as carbon monoxide, should not be used. We have drawn attention already to the dangerous action of even minute quantities of CO in rendering explosive mixtures of coal-dust with small quantities of fire-damp. Again, those explosives, as gunpowder, of which the products of combustion are of a highly combustible nature, should not be employed.

Where the explosive burns instead of explodes, which, taking into consideration the enormous amount of explosives used in mines is, however, a comparatively rare accident, there is a large proportion of CO and nitrous fumes evolved, and gas-poisoning on a large scale may occur; as in South Africa where ten men lost their lives, and at Kalgoorlie, Australia, where six men perished. The term "*Misfire*" is used in the Transvaal where, instead of there being complete detonation, a partial explosion takes place; where, in other words, there is a slower form of combustion. This fact cannot be insisted on too strongly, viz.:—that whenever detonation is imperfect, CO gas may be formed in dangerous amount. Besides the explosive burning or imperfect detonation, the detonator may become separated from the charge, which in consequence fails to go off. Experts find it difficult, however, to point to the cause of this failure to explode. The Transvaal Miners' Phthisis Commission heard a great deal of expert evidence regarding this matter, which is a serious question in South Africa

and it concluded that the following were the principal conditions which might produce it, viz. :—bad quality of explosives, or their deterioration in quality owing to their having stood too long or to their having been stored in an improper place ; bad quality of detonators, or weak or damp detonators, as where they have been exposed for some time in moist workings ; "also overcharge of explosives in proportion to load" (page 17). Where the explosive burns instead of detonates there is a characteristic smell produced.

Dr Haldane made a number of analyses of the air of "ends" after blasting with gelignite, and in every case found small percentages of CO present, viz. :—0·02 per cent., 0·05 per cent., and 0·065 per cent., and also very minute quantities of nitrous gas. He pointed out that, although the percentage of CO in most cases was very small, more than 0·1 per cent. would often be met with in blasting with this explosive. Where other explosives are used, much greater percentages of CO may result. A point established in the analysis in the air of mines after blasting operations is the small proportion of nitrous gas present, indeed so small as to be of no account. Dr W. C. C. Pakes, who gave valuable evidence before the Transvaal Commission regarding the action of noxious gases in mines, held that the irritation of nitrous gas was less than that caused by the dust, and that the danger, compared with CO in the porportion usually present in mines, was almost negligible. Laymen, however, lay great stress on the poisonous character of nitrous fumes, but this is owing to the pungent and irritating action of the fumes on the mucous membrane of the throat, etc., which tends to mask the less noticeable but much more deadly CO. Dr Pakes also drew attention to the fact that chronic poisoning by the fumes from explosives must be very common, and that this would have a serious effect in reducing the resisting powers of the body, more especially of the lungs, to disease, and might lead to the development of Silicosis, Pneumonia, Meningitis, etc.

CHAPTER V.

Causes of Death in Colliery Explosions.

THIS sketch of the principal causes of death in explosions is drawn mainly from the official reports of many explosions both at home and abroad, and especially from Haldane's work on "Causes of Death in Colliery Explosions and Underground Fires," the writings of Brouardel, Dr Firmin Dervieux's Report on the terrible Courrières disaster where 1,100 workmen perished, the recent articles by Dr Llewellyn on the Darran explosion, those by Dr Hatton on the Hulton disaster, and from many other sources.

Deaths in explosions may be caused in several ways :—

(1) By violence—the force, direct and indirect, of the explosion, falls from the roof, etc., brought about by the explosion.
(2) By burns caused by the flames.
(3) By after-damp (Carbon Monoxide).

The following facts from two recent colliery disasters, in which the loss of life was terribly heavy, will give a fair conception of the causes of death in most of the cases. They are taken from the reports of the Whitehaven disaster by Dr Harris, and from that of the Hulton disaster by Dr Hatton. In passing, it may be said that we find ourselves in agreement with Dr Hatton's recommendation that skilled pathologists should be present after each great mining disaster to make a thorough examination of the bodies, because in previous disasters the medical reports regarding the causation of death were found to be erroneous; that is to say, the causation of death in many of the cases, owing to the bodies being covered by caked dust, was attributed to burning, when it was undoubtedly due to CO gas. In the Whitehaven disaster, Dr Harris examined 133 bodies. Of these he held that in all probability 85 died from CO poisoning; and that 41 showed signs of burning, of which, however, only 30 exhibited evidence of severe burning. Distributing these in detailed causes of death, his opinion was that :—

12 were killed outright by the violence of the explosion;
35 were "undoubtedly poisoned by CO";
38 were suffocated by smoke or other fumes, aided possibly by CO poisoning;
12 by smoke-fumes and probably CO poisoning;
1 died from shock or suffocation;
30 were killed by burning and shock;
1 (calcined remains) by burns, or shock, or suffocation; and
4 by the combined effects of burns, shock, and suffocation by CO.

133

Dr Hatton examined 343 bodies after the Hulton colliery disaster, of which :—

224 deaths were caused by CO ;
53 deaths by force of the explosion ;
63 deaths by force of the explosion and CO ;
2 deaths by asphyxia ;
3 unstated (probably by explosion and CO) ; and
1 by shock and burns.
———
343

Of the total dead bodies 312 were found uninjured ; 92 were charred and scorched by flame ; 31 were injured by violence, including those found under falls. Of the 343 bodies 19 remained unidentified.

I.—Deaths due to Direct Violence.

A certain percentage of deaths, generally small and varying from 11 per cent. to 18 per cent., is caused by the direct violence of the explosion and also by falls. Of the 343 cases at the Hulton disaster 15 per cent. were killed by violence, and in 34 per cent., besides signs of violence, there were also evidences of CO poisoning. Mutilated bodies showing signs of extreme violence, being shattered and cut to pieces in some cases, may be found in different parts of a mine wherever an explosion has taken place ; because in every large explosion, as Sir H. Hall has pointed out, several explosions occur almost simultaneously or with a fraction of time between them, so that the blast appears almost as one single explosion. Plans[1] are now always drawn up after an explosion showing where the various bodies were found and describing how death took place. It has already been noted that when an explosive gaseous body is ignited, the force generated increases up to a maximum and then rapidly diminishes in strength, so that there are intervals of distance where there is no mutilation or dismemberment of the bodies when these have not been exposed to the direct violence of the explosion. Outside the zone where the force is greatest, the clothes of the victims may be ripped open or torn away, or the men may be pushed along without any further damage being done them, showing that the blast has been spent considerably before reaching them, while at a point even further away the explosion may be felt only as a slight gust. In an explosion in New Zealand, for example, the explosive blast removed every stitch of clothing from a boy, but the body itself showed no signs of burning or injury.

In the Hulton disaster 312 out of the 343 bodies showed no signs of disfigurement ; and in the Albion disaster 290 bodies were found scattered along 9,000 yards of road, fifteen of these being terribly mutil-

[1] See Appendix.

ated and burned, while the majority (275) showed no traces of violence. These fifteen bodies were not found together but separately at different points, which indicated that there had probably been a series of rapid explosions and not one continuous blast. The conditions of the roads where the mutilated bodies were found showed the same signs of violence, as evidenced by shattered doors and hutches and extensive falls of débris, etc. In one place a body was found with the leg torn off at the knee, and the head lying three or four yards from the body. At another place a group of six bodies, much mutilated and battered, was found; at a third, the body of a man much disfigured and burned, with a leg missing; while at other places some of the bodies had been blown to pieces, head and legs being torn from the body and nothing left but the trunk. In the Hulton disaster, ten of the 343 bodies showed terrible injuries; "in four nearly every limb was fractured, the skull torn off, brain absent, and chest and abdomen laid open; and in one case the liver and heart were laid upon the ribs. One man was blown bodily about forty yards, and then fell; another about 135 yards"; and so on.

At the Monongah disaster, perhaps one of the most awful which ever took place in America and in which 340 died, although the bodies were distributed over a few miles of road, it was found that mutilated bodies discovered in the wrecked parts were limited to a very small zone, and that these wrecked zones were in different sections many hundreds of yards apart. In the recent Whitehaven disaster, also, the bodies in No. 3 district alone showed signs of violence or of burning. In fact, that is what is generally found in most explosions, viz.:—places where there are evidences of extreme violence succeeded by lengths of road where movable materials are not disturbed, no damage is done to the roads, and the men are uninjured.

In the account by Firmin Dervieux[1] of the Courrières disaster, there is a most detailed, graphic, and gruesome description of the awful injuries found, which he vividly describes as "a veritable dance of death."

The velocity of the volume of air needful to hurl men, hutches, and all movable articles in the pit to varying distances, to blow down permanent stoppings many feet in thickness, to tear up pavements and cause tremendous falls measuring yards in length and weighing hundreds of tons, must be enormous. This column of air itself acts just like a violently-propelled projectile, and produces the horrible and extensive mutilations found after great explosions. Many of the injuries may be explained by the bodies themselves being used like missiles and literally blown against the walls, roof, and pavement. Sir James Hector,[2] writing on the Brunner mine disaster, where all the men working in the mine,

[1] Firmin Dervieux: *Annales d'Hygiène Publique*, Nov. 1906, p. 533.

[2] Sir J. Hector: Trans. and Proc. New Zealand Inst. M.E., 1896, Vol. XXIX., p. 602.

sixty-five in number, died, described the body of a man which had been propelled or driven up an incline of 300 feet in length and there been smashed against the wall of coal. At the top of another incline the body of another man was also found smashed to a pulp. Again, the wrecked woodwork, hutches, props, etc., hurled with terrific force against the men, may also account for many of the lesions; thus might limbs be carried away, bodies decapitated, thoracic and abdominal cavities burst open, and so on.

Dervieux attempted to account for the lesions by violence found on some of the bodies which were immediately in the zone of the explosion. He likened the passages of the mine to the barrel of a gun, the explosion playing the part of the cartridge, and the miners filling the office of the bullets: the men were thus violently projected against the walls, on which their bodies were literally reduced to pulp.

Others may be killed by falls of roof, owing to the supporting timbers being swept away and the roof giving way, or many yards of the roof, etc., may be directly brought down by the violence of the concussion. In all those directly killed by violence at the Courrières disaster and examined by Dervieux, no CO was found in the blood; indeed, had these survived any time, CO would have been inhaled and its presence therefore been demonstrable. This is a point which should always be proved before the death is put down to direct violence.

It has long been recognised when an explosion occurs, that there is first of all an enormous expansion of the air as a result of the production of hot gases. This is followed by a shrinking—the so-called recoil—brought about by the contraction of this expanded mass of gas by cooling, and by condensation of the steam formed. The result is that, owing to the partial vacuum formed at the site of the explosion, the air at some distance, which has either been compressed or forced along the path of least resistance, rushes violently back.

Dervieux divides the wounds by violence into two classes: the *first* comprising wounds produced from *without inwards*, due to the *enormous compression* of the gases at the time of combustion; the *second*, wounds produced from *within outwards* during the recoil or cooling of the gases. The latter are found only on bodies at a short distance from the explosion in a cul-de-sac or gallery closed by falls. It was only in this way that Dervieux was able to explain the bruising and the many injuries from without inwards, and the bursting to pieces when the wounds were produced from within outwards. This helps to explain the bursting open of the closed cavities of the body, thorax, abdomen, etc., instances of which are to be found in some bodies in large explosions.

Position of the Bodies found After an Explosion.

For a number of years past it has been the custom of Inspectors of

Mines after an explosion to mark on their plans the spot where each man was at work, along with his number, and also the point at which each body, which is numbered and afterwards identified, was found. One can easily appreciate this to be a most important proceeding, as from such a plan much information may be gained regarding what the men were doing at that time, the causes of death, the prevention of the same in future, and the interpretation of facts regarding the manner in which the explosion had been started. (See Plans, etc. in Appendix).

It has already been stated that the mutilated bodies are generally found where there are evidences of great damage done to the surroundings. Most of the deaths, however, are perhaps due to CO poisoning. This may be rapid or gradual. In cases of rapid poisoning many of the bodies are found in the identical positions in which they were at the moment. Some of them, for example, are found with the food they were eating in their hands, others while putting on their clothes, and others with their picks or other implements in their hands in an attitude of work. These may be taken as cases of acute poisoning. From the appearances of the bodies found after the Courrières disaster, there was nothing which pointed to the onset of convulsions before death, a fact which bears out the statement made by Brouardel that convulsions are seldom seen in poisoning by CO. This statement is not supported, however, by all observers.

After a wide-spread explosion there are graphic pictures, in the positions of the bodies of men found, of the circumstances in which the deaths took place. For example, along the main roads many bodies of men, who were undoubtedly struck down as they tried to escape, are found. Suspecting that something was wrong with the air-current, or owing to the noise or blast of air, or perhaps feeling "queer," they were probably proceeding to make their way into the main roads, but the atmosphere becoming worse, or rather because of the CO accumulating in their blood, they gradually became more and more affected and more helpless, and staggering and tottering they fell forward on their knees, their heads dropped to the ground, and the poisonous gas continuing its deadly work, they have died in this position; or, they may have felt giddy and helpless and leaned up against the walls for support, when death overtook them. In not a few cases, where fire has followed the explosion, it is found that some of the men, to save themselves from the irritant fumes, have placed their handkerchiefs over their mouths. At the Whitehaven disaster, for example, a number of dead bodies were found with a handkerchief or scarf bound over the mouth.

Sometimes instances are found where men, knowing the poisonous nature of after-damp, have tried to build stoppings, which they have attempted to render gas-proof with their clothing. In some cases this

has proved successful; but at Courrières, where fourteen men tried to shield themselves in this way, it proved of no avail, as they were quickly killed. This was indicated by the untouched stores of food beside them.

Table showing position of bodies found in three great colliery explosions:—

TABLE XV.

Disaster.	Intake Roads.	At the Face.	In Return Air-ways.	Total.
Seaham, 1880 . .	116	1	44	161
Llanerch, 1890 . .	136	27	11	174
Albion, 1895 . .	194	76	6	270

Haldane has made a most careful inquiry into the positions in which bodies are found after explosions. This inquiry shows that, as a general rule, the bodies are found along or near the track of the explosion. Many of them are found close together on the main roads. Evidently these are the bodies of men who have come from the working face, and have been making their way to the shaft when they were overcome by after-damp. These men, in trying thus to escape, simply ran into danger. From information gathered from many explosions, it may be said with confidence, that in many cases men might have escaped had they remained at their places till the after-damp in the roads had become diluted and dispersed. But these men, in hurrying along the main roads, not only ran the certain risk of poisoning by CO, but also of being injured or killed by falls from the roof. It is the same in underground fires,—the men rush along the intake roads to the down-cast and right into the poisonous air. The following interesting point was brought out in the West Stanley disaster where 167 men perished, viz.:—that in the side-tracks off the main roads, air might have been found in sufficient quantity and of sufficient purity to support life. In this disaster, the fact was that the men who left the main roads escaped, while those who went by the main roads were poisoned. The men who were rescued from the Courrières disaster after so many days saved themselves by retiring into the working-places. In the Parkslip disaster it was proved that all the workmen, fifty-six in number, in one of the sections, would have been saved had they remained in the workings till the after-damp in the intake and return drifts had cleared away sufficiently, as the eighteen were who eventually escaped from these workings.

II.—Burns.

In small local explosions, which are of frequent occurrence in fiery pits and which result in a few men being injured or killed, not infrequently the most serious injuries are those caused by burning. Although these burns are always more or less superficial, they may cover such large areas of the body as to be very serious. Many die from the combined effects of the shock and burns. In the Prussian Fire-damp Commission's Report, particulars are given of fire-damp explosions from 1861 to 1884. The Report takes count of 346 fatal explosions with 916 killed and 447 injured, and of 939 non-fatal explosions with 1415 injured; that is to say, in 1285 explosions there were 916 killed and 1862 injured. Of these, 839 were killed by the immediate consequences of the explosion, that is, by burning and crushing, or from injuries received at the time; 287 or 25·2 per cent. were killed by the after-damp; and eleven or 1 per cent. perished during the work of rescue, and mostly by CO poisoning.

In large explosions, which are mainly caused or propagated by coal-dust, it was formerly thought from the charred appearances of many of the bodies, that the deaths of these persons resulted from burns. Even in recent explosions the like mistakes have been made, doubtless owing to careless examination of the bodies. For instance, at the Parkslip disaster in which 112 men met their deaths, the doctors who examined the bodies gave it as their opinion that 100 deaths were due to burns and shock, but the evidence of Atkinson, who has had an unrivalled experience of explosions, and of others, showed that the bulk of these men could not possibly have been touched by fire, for they were in parts of the mine not reached by the flame, and they were alive for hours after the explosion, though ultimately dying of CO poisoning.

Upon all the 343 bodies of those who perished at the Hulton disaster which were examined by Dr Hatton, there was a thick and uniform coating from head to foot of coal-dust which could quite easily be washed off.

It has been estimated that the temperature of an exploding mixture of methane, coal-dust, and air would reach 2000°C, but that this would last only momentarily, as much heat would be absorbed in heating and distilling the dust already suspended in the air (Haldane); and the temperature would very rapidly be brought down by contact with the cold walls, etc. Now under the action of a flame of great magnitude and of such intensity, the bodies of men and horses would be incinerated, and all timber burned and charred. But at most explosions, unless where the explosion has given rise to a secondary fire, the wood shows little evidence of the great effects of heat, and only in certain parts are the men found slightly burned and covered with coked coal-dust, and

the hair on the head and face merely singed, not burned into the roots. Generally the burning is not extensive, because when the explosion has passed over long roads, the oxygen is consumed and further combustion is consequently arrested till the return of fresh air, which is often prevented by falls from the roof, breaking down of doors, the destruction, perhaps, of the fan, and other derangements of ventilation caused by the explosion. Indeed, even where the flame has passed, it could only have been of short duration, because in many cases it is only on the exposed surfaces that burns are found. In the Hulton disaster, out of the 343 bodies, 92 were found to be burnt or scorched. In only two of the cases was the burning deep; in the others, although it was extensive it was not deep, the hair of the head in all these cases being burnt to within half an inch of the scalp. In two cases there was charring of the skull, "and in many places in the upper part of the body and limbs the skin was hanging off like scorched rags." Dr Hatton drew attention to the fact that in nearly all the cases of burning it was the upper part of the body only which was affected, for even when the lower part was uncovered it did not appear to have been touched by the flame, showing unmistakably that the flame had passed along the upper part of the gallery and not along the pavement. It was very seldom that the clothing of the lower limbs was found burnt. In only one case in this disaster was the body badly burned or charred, the leg in that case being burnt to a cinder.

Superficial burns, then, are met with in a certain percentage of cases along with singeing of the hair, the burning covering a more extensive area where the skin has not been protected by the clothing, the exposed parts being covered by a thin layer of adherent coal-dust. Some of the bodies in the Courrières disaster, and in many other explosions, looked as if they were charred. Careful examination of these, however, proved that there was no real charring, but that the skin was thickly encrusted with fine coked coal-dust which had become adherent to the skin. Brouardel[1] has also drawn attention to this. He says: "the burns, then, are not very serious in general; but as the air holds a great quantity of coal-dust, and this dust becomes encrusted upon the skin of the men, the bodies become quite black, and seem to have burns of the third degree." When this crusted material is rubbed off, the epidermis may come away; but the deep skin is not injured. Brouardel and Haldane hold that this loosening of the epidermis is not the result of the formation of blisters, but is due to the sudden development of gas and vapour consequent on the application of intense heat momentarily applied.

Superficial burns of the conjunctiva, cornea, and mucous membranes may be met with. Dervieux carried out *post-mortem* examinations on horses which had been burned. He found that from the nostrils down to and including the bronchial tubes, the mucous membrane was covered

[1] Brouardel: *Les Explosifs et les Explosions*, Paris 1897, p. 63.

with coal-dust under which at certain points were found little ulcerations. The lungs were extremely congested, and contained some extravasations of blood with centres of pulmonary apoplexy. The latter condition, however, was more likely to be caused by the CO than by intense heat. Haldane, in his examination of horses, found nothing except slight scorching, and this did not pass beyond the lips and tip of the tongue, the inner surface of the bronchi being quite normal. None of the survivors showed definite signs of internal burning. The trachea showed a little coal-dust, and was slightly congested owing to the inhalation of irritant gases. When discussing the subject of pneumonia following CO poisoning, we shall have more to say about the lesions in the respiratory tract caused by burns.

After great explosions very few cases are found in which the body has been acted upon by flame for any length of time. Corpses have been found in the pugilistic attitude, "dans la position de combat," as first described by Devergie, and as figured by Brouardel in his book on Gas Poisoning and by others. This is due to coagulation of the albumens from roasting of the muscles by the action of gases at a very high temperature. Dervieux found a few cases in the Courrières disaster. The clothes were intact but the boots were cracked; the skull and thoracic cavities were burst open; there was shrinking of the limbs with retraction of the skin; and opalescent cornea: indeed, altogether very like the picture drawn by Brouardel of his cases at the fire at the Opera Comique. Sometimes amputations of limbs are found. These, and the bursting open of the cavities by heat, differ entirely from the mutilations produced by the force of the explosion.

It is necessary to differentiate clearly between the lesions found on the bodies of those who have died from their burns, and those in which the dead bodies have been acted on by the fire, and, again, in respect of dead bodies which show signs perhaps of extensive burning but where death is due to CO poisoning. It was conclusively proved, for example, at the Courrières disaster that some of the men were extensively burned, that these burns indeed would undoubtedly have ended fatally in time, but that death was actually produced by CO poisoning.

Even in cases of survivors where there is extensive burning, the men complain of very little pain, so benumbing is the effect of the CO and other gases which they have inhaled. In fact in some cases it is hours after the explosion before they recognise that they have been burned. Dr Hatton insists that at the Hulton disaster in only one case was there any indication that the burns had caused much suffering. In this case the clothing had caught fire, and the man had taken off his boots in order to pull off his trousers, these being found burnt to ashes, while the body itself was found about fifty yards further on. Dr Hatton is of opinion that in this disaster in no case was death due to burning.

CHAPTER VI.

SYMPTOMS OF POISONING BY AFTER-DAMP AND FUMES FROM EXPLOSIVES.

Poisoning by After-damp, Gases from Explosives, etc.

It is interesting at the outset to remember that Thomas, one of the greatest mining authorities, wrote on this subject as follows:—"The fatal effects produced by after-damp have long been an enigma to practical miners." The direct effects of the explosion, that is the deaths by violence, were easily explained, and many of the deaths by suffocation were accounted for by authorities as due to the using-up of the oxygen and production of CO_2, which with the remaining nitrogen was sufficient in the absence of oxygen to cause suffocation. But this could not explain the deaths which occurred in places very far distant from the explosion where there still remained a certain amount of air. The fact was also pointed out that in certain instances lamps were found burning beside the dead bodies of miners. Thomas[1] was, perhaps, the first to suggest that CO gas was the poisonous agent in after-damp. "During every explosion" he wrote "large quantities of this gas, CO, are formed, and the fatal effects of after-damp are in great measure due to its presence." Also as regarding the importance of coal-dust in the production of CO, he further wrote:—"From experiments carried out by the author it would appear that CO would be generated at certain points owing to the presence of coal-dust, and if a cloud of coal-dust was raised, as is frequently the case, carbonic oxide would be formed, and this gas would consequently render the after-damp very poisonous in cases where the force of the explosion was anything but formidable."

Symptoms.

The symptoms of what might be termed gradual poisoning by CO, such as are met with after an explosion, are well known from the experiences of survivors and of members of rescue parties, as well as from the experimental work of Haldane and others.

Haldane's Experiments.

Haldane found when the blood has absorbed CO to the extent of 20 per cent., that symptoms appear only on exertion, and consist mainly

[1] Thomas: "Iron," Feb. 1875. *Coal, Mine-Gases, and Ventilation*, p. 154.

of slight giddiness and shortness of breath. The first decided symptoms during rest make their appearance only when the blood is saturated with about 30 per cent. of the gas; that is to say, when about one third of the oxygen-carrying red blood corpuscles are thrown out of action. With every increase up to 50 per cent. of saturation the symptoms become more and more imperative; at this point staggering begins, and on the slightest exertion the person loses consciousness. Death occurs when the saturation attains about 80 per cent. This observer has found in many bodies after explosions, that the blood was saturated by CO gas to this percentage.

Some very important practical points have been brought out in Haldane's experimental work. The first, a point first described by Gréhant is, that although there may be a very small percentage only of CO present in the air respired, the action of the gas is cumulative, and that the CO is gradually but continuously taken up by the hæmoglobin, and displaces the oxygen till the body suffers from oxygen starvation. Although there may be only 0·1 per cent. of CO present in the atmosphere, therefore, the blood can be saturated in time up to 50 per cent., and at this point dangerous symptoms present themselves in the loss of power over the limbs and staggering and unsteady gait, which may be the first signal, an indication presenting itself perhaps too late to allow the victim to get out of the dangerous atmosphere. Even after prolonged exposure to 0·1 per cent. of CO recovery is, however, general. With 0·2 per cent. cerebral symptoms would appear in the form of loss of consciousness and absolute loss of power, and, in course of time, death would follow. Exposure to 0·3 per cent. proves more rapidly fatal.

The same observer has also worked out the time necessary to produce dangerous symptoms with different percentages of CO. This has a most practical bearing on rescue work in mines, etc. In an atmosphere containing 0·1 per cent. of CO, it would take a man at rest $2\frac{1}{4}$ hours to develop 50 per cent. of saturation, but when walking, just about one hour. Thus men engaged in rescue work in an atmosphere containing 0·10 per cent. of CO would begin to be affected by unsteadiness of the legs, and perhaps by loss of power, in the course of an hour, and even earlier if they had much climbing over "falls" or were making great exertion in attempting to carry an unconscious man. Where there is 0·20 per cent. of CO present, half an hour would be sufficient to produce these symptoms; and with a percentage of 0·30 per cent., twenty minutes. Such experimental work as Haldane's is most valuable, as it indicates the great danger to which men are exposed in rescue work. In rescue work men may have proceeded a considerable distance into foul air before becoming affected, and have not sufficient oxygen-carrying hæmoglobin left to bear them back to safety.

With regard to CO poisoning in mines, the process generally is one of gradual poisoning by small percentages of CO. There is seldom what the French call "intoxication massive," which is so often found in poisoning by furnace and producer-gas.

It is well to insist on the insidious nature of CO poisoning; indeed the men may become unconscious before they have really comprehended that there is anything out of the ordinary wrong with them; and the peculiar dulling influence which this gas has on the brain helps to produce this effect. In some cases poisoning takes place very suddenly, and after many great explosions, *e.g.*, the Courrières disaster, many of the men are found dead in their places, with their picks in their hands as if working, and others as if they had been taking their food when overtaken. To judge from the placid expression on their faces, death must have been not only rapid but painless.

It would seem that the peculiar action which CO, when inhaled in large percentage, has upon the brain, and which leads to wild excitement, intoxication, shouting, and other mental symptoms of exaltation, is not nearly so common in poisoning in mines where the percentage of CO present is much smaller. Indeed most evidence points to the gradual process of poisoning as having generally a benumbing or deadening action, an effect which develops slowly and insidiously.

The following is the story generally obtained from men who have been overcome when building-off a "waste-fire," or from those who have returned to their places too soon after shot-firing, more especially if blasting powder or certain kinds of gelignite have been used, and where the ventilation is not very good, as in "driving through" a connection where, of course, there is no through ventilation.

They first experience sensations of giddiness or swimming, feel queer, with perhaps ringing in the ears, sparks before the eyes, fluttering or throbbing of the heart, and slight breathlessness when they are making any severe muscular effort. If they understand from these symptoms that they are breathing dangerously tainted air and come out of their places into the main roads where the air is much better, these sensations quickly pass off, leaving perhaps only headache, more or less severe. But if this warning be neglected (in some cases there may be no preliminary warning), the symptoms become more marked, the miner feels that his legs are beginning "to go from under him," and he totters and staggers about till he sinks to the ground in a semi-conscious or unconscious condition.

This is not the only manner of onset; unfortunately in many cases it is much more insidious. They may feel as if they were thoroughly worn out, or may experience a feeling of lassitude which passes into a dangerous drowsiness, the senses gradually becoming clouded, until at

last they become unconscious. A good example of this action is furnished in the case of Dr Turner, who was decorated by King Edward for heroism during rescue work at the Darran disaster. He had no symptoms until he exerted himself in trying to rouse his companions; he then felt tired; wanted to lie down; fell down several times; and finally lost consciousness. Or again, as some of them have told us, a miner may automatically go on with his work, feeling something wrong, and yet be actually unable to stop and consider what the cause really is. In all these cases, unless the men are quickly succoured, death may take place.

Fortunately, however, in the cases which we are considering, the gassing takes place in a localised zone, and as the men are seldom alone, their mates quickly drag them out of harm's way. These cases do not recover so quickly as those first described, although by the time the patient arrives at the pit-bottom, where he has probably been run on a hutch, he may have considerably recovered. But during the walk home—and the miner always insists on walking home if his legs can bear him—the exertion frequently brings back the palpitation and throbbing of the heart and main vessels, with breathlessness, feeling of swimming, staggering gait, and generally, also, nausea, vomiting and retching. The headache may be very severe, each step increasing the throbbing feeling in the temples and forehead, which the man supports and compresses with both hands. With rest he generally feels a great deal better, and he may be able to resume work in the course of a few days.

The general story given by members of rescue parties after explosions in mines is that, after going a certain distance in the poisonous air, they begin to feel weak, to become a little giddy or drowsy, and perhaps to stagger slightly on walking. When they have to make increased exertion, as for example, in passing over a fall, or in lifting one of the victims, these symptoms became much exaggerated. Some, indeed, feel that they are quite incapable of making any extra exertion. There is also palpitation of the heart on exertion, and a feeling of fulness or of actual pain in the head. Some just before losing consciousness complain of a feeling of swelling of the head. Other symptoms which are sometimes complained of, more especially in underground fires, or where a fire has resulted from explosion, point probably to the presence of a small quantity of sulphurous acid. Among these are dryness of the throat; a gripping sensation at the throat; a feeling of great thirst; and smarting of the eyes. In poisoning by gases produced by certain explosives when they burn instead of detonating, nitrous fumes often cause severe coughing. In other cases the men may sink helpless with very little preliminary warning.

After some great explosions, graphic pictures of the gradual poisoning

and the gradual loss of power over the limbs may be observed, many bodies being found along the main roads in most suggestive positions. Brockmann, whose work is referred to in the Report of the Austrian Fire-Damp Commission, relates interesting cases of surviving miners who were poisoned by CO from pit fires, and who declared that they had a curious feeling as if their hands, arms, head, and whole body had suddenly become swollen, and that when ascending the ladders (this was in the year 1854) they felt the rungs and sides of the ladder as if they were two or three times their usual size. They also declared that they felt as if they were walking on a soft, yielding substance.

After-effects of Poisoning by CO in Mines.

Perhaps the commonest of all symptoms complained of after exposure to CO is *headache*, which may come on in paroxysms and be very severe; the pain may be felt either in the temples, when it is often of a throbbing character, or in the forehead, or eyes, or more rarely in the occipital region. A considerable percentage of sufferers complain of a feeling of constriction. For example, nearly every one of the sixty miners who were exposed at Wanlockhead to the gases from burning timber in the lead mine complained of violent headache and a feeling of constriction in the temples. In some the throbbing was so exaggerated as to be agonising, the patient feeling as if his head were about to burst open. The pain is always increased by movement, and we have seen men, who have walked home after being gassed in mines by the fumes from explosives, holding their heads with their hands and complaining bitterly of the pain. Where headache is not complained of, there may be feelings of distension in the head without actual pain. Sometimes the headache does not come on till the patient has reached the open air.

With the headache there may be ringing in the ears, and there is frequently some interference with vision which may become dim, indistinct, and blurred, and the patients complain of not being able to make out the details of anything before them. With the severest forms of headache the patient may complain of flashes of light. In rare cases hallucinations of sight have developed, as also blindness, which, however only lasted for a few days. In others, pain in the eyes of a constant boring, aching character has been a prominent symptom, while some have declared that their eyes felt as if the balls were about to burst from their sockets.

Giddiness may be very marked, more especially if the patient makes any exertion. This usually disappears or becomes much less if the patient is kept lying with his head low, as it is produced by disturbances in the cerebral circulation. Some can only get rid of this feeling by keeping their eyes shut, for whenever they open them the giddiness returns.

Powerlessness.—We have already drawn attention to the characteristic effect which poisoning by CO has on muscular co-ordination and muscular power of the lower limbs, which ranges from a feeling of weakness and staggering up to complete impotence. We had a patient who was engaged in driving a road through to meet another place so as to complete the circuit of ventilation. He had fired a considerable charge of gunpowder, and soon afterwards went back to his place. In a very short time he felt as if his legs were paralysed; he felt dead from the pelvis downwards. His mind, however, was sufficiently clear to understand his danger, and he managed by the use of his arms, which fortunately retained their power, to drag himself into purer air.

In such cases, then, the patient loses control of his limbs and staggers about when put on his feet, or he may collapse, his legs giving way altogether. The hands and arms may also be affected, the patient losing his power to grip. In one case which came under our notice, where a man was poisoned in his working place by CO which had found its way through fissures from a built-off fire, the arms were affected before the legs. The man felt his pick becoming heavier and heavier, and his arms more powerless till they failed him altogether. Soon after, the lower limbs began to be affected, and when he got on his legs he staggered about in a helpless manner. Sometimes there is a remarkable loss of control over the muscles of the neck, and in one case which came under our notice, although the patient was able to sit up, the head rolled about in a particularly helpless fashion. With the loss of power, complete or incomplete, there may be loss of sensation in the hands and feet, a feeling as if these members were quite dead.

Cases have been met with where there were burns of considerable severity, and yet the patients were quite unaware of their existence. The hands and feet may become quite cold and white, and this condition may extend up the arms and legs. There may also be prickling sensations. In one well-marked case in which powerlessness and loss of control over the limbs were present, there were also backache, pains darting from the back into the legs, a feeling as if the legs from the knees downwards were dead, prickling and tingling sensations in the legs and feet, principally the soles, and frequent attacks of cramp in the muscles of the calves. For a long time the patient had no proper control over his limbs; indeed there was sometimes sudden loss of power, and the patient fell to the ground. Dr Llewellyn[1] reports that in one of the survivors of the Darran explosion there was a feeling of weight in the back, and also of weakness and heaviness in the legs.

This condition generally passes off very quickly, but we have seen

[1] Llewellyn: "The Darran Explosion from the Medical Standpoint." *B.M.J.* 11th June, 1910.

cases where it remained for several months, the staggering gait coming on whenever the patient was fatigued by excessive walking.

Yawning.—This is Nature's mode of assisting the aeration of the blood, and is not uncommonly seen after CO poisoning or after exposure to black-damp. It may, indeed, be very exaggerated. Dr G. H. Logan, of Cleland, had one case in which a man who suffered from CO poisoning through having returned too soon after the firing of a heavy charge of blasting powder to a place where the ventilation was deficient, yawned every few minutes for two and a half hours, and at frequent intervals thereafter during the rest of the day. His jaws became very painful from their unwonted exercise.

A patient after gassing is generally dazed and stupid-looking, with little or no control over the mind, and he often feels drowsy and sleepy.

Vomiting is another very common symptom of gassing. It may be very severe and accompanied by retching. It may also be accompanied by severe abdominal pain. All the survivors of the Courrières disaster[1] complained of pain about the umbilicus. Some of them also vomited blood, and two had *diarrhœa* with blood in the motions. At the Crarae disaster[2] already alluded to, of a number of persons who were overcome by the fumes, one or two who were treated in hospital vomited blood. We have on several occasions seen both vomiting and diarrhœa in miners who have been exposed to black-damp; and we have also a patient who has been repeatedly overcome by gas when cleaning the flues at a Steel Work, and who declared that instead of having vomiting and pain in the stomach after being gassed, these being commonly recognised symptoms, he had diarrhœa with severe pain about the umbilicus. Brouardel[3] also found among the survivors of the fire at the Opera Comique, several cases of intestinal hæmorrhage.

Obstinate *Hiccough* is a symptom to which Josephson[4] called attention in connection with CO poisoning during blasting operations. A remarkable case in which hiccough was the most prominent symptom, recurring at very short intervals for three days till death ensued, is reported in connection with the Courrières disaster.[5]

Marked *Shivering*, feeling of cold, and inability to keep warm are frequently complained of; this condition may last for a few days, and in some cases for weeks. A man whom we examined, who was poisoned by CO from a fire in a mine, had recurring attacks of severe shivering for a week. This must be distinguished from trembling and tremors. After minor cases of gassing in pits, the patient may generally be found

[1] Firmin Dervieux: *Annales d'Hygiène Publique*, 1906, p. 542.
[2] Crarae Disaster. Report by Col. Ford.
[3] Brouardel: *Les Asphyxies par les Gaz.* Paris, 1896, p. 295.
[4] Josephson: *Annales d'Hygiène Publique*, 1860.
[5] Firmin Dervieux: *Loc. cit.*, p. 538.

for a few days sitting in a drowsy state by the fire constantly complaining of chilliness. This loss of heat is attributable to interference with the heat-producing centre, diminished conduction of oxygen to, and therefore defective oxidation in the tissues, and also to the action of CO on the heart and blood corpuscles.

Palpitation of the heart and oppression about the chest and præcordial region with breathlessness are often met with. Exertion, as in walking home or in climbing over a fall, makes the condition worse. These symptoms generally pass off very quickly, although in some cases they reappear at an interval of days after the gassing if the patient over-exerts himself. When these attacks supervene some patients have a feeling of impending death, and experience darkness before the eyes and booming in their ears. Their legs may at the same time give way and they may fall to the ground. The palpitation is often severe and lasts for a few days; and it is generally worse at night, so that the patient's rest is much broken. With the palpitation there may be evidences of vaso-motor disturbances, as seen in attacks of flushing of the face, head, and neck, followed by cold sweats.

It is often found among men who have been gassed and rendered unconscious while engaged in "building-off" underground fires, that when they are brought into the main roads they quickly come to consciousness, only to relapse, however, into unconsciousness when they are taken into the open air. We have already drawn attention to the fact that certain symptoms are aggravated whenever the patient reaches the open air. The same has been frequently found amongst survivors from explosions and members of rescue parties who have been overcome by after-damp, on their being brought to the surface. The exposure to the colder atmosphere frequently causes them to become worse, and even to lose consciousness, while in a few instances fatal results have ensued. In a less degree we have found that men who have been working in air heavily contaminated with gas (*e.g.* in a steel shop where, owing to a leakage there was a considerable percentage of gas present), experienced practically nothing wrong till they had occasion to go outside, and then they felt their heads begin to swim and their legs to become weak. The fact of exposure to cold causing relapses and intensifying serious symptoms has a practical bearing on the treatment, and we have no doubt that lives have been lost which might have been saved had this been realised and suitable preventive measures been taken.

Tremor is frequently seen after CO poisoning in mines. It is generally found, for example, in the hands when they are extended, or in the arms, head, or legs when in certain positions. There may be a general trembling of the whole body which may last for a few days. In some of the miners in the Wanlockhead disaster these muscular

tremors remained for many weeks. In one survivor of the Darran explosion, who was unable to resume work for a considerable time owing to great nervous prostration, inability to perform any physical exertion, loss of appetite, etc., there was marked weakness of "grip," with tremors of the hands; and in another, who complained of weakness after the least exertion, there was pronounced tremor of the hands, and well-marked nystagmus, which set in immediately after the accident, although there had been no previous trouble with the eyes. We shall describe under illuminant-gas poisoning cases by Becker and others, where the tremor was so marked as to simulate disseminated sclerosis. The shivering, which is so often found after gassing, must not be mistaken for tremor, nor where it is very exaggerated, for epileptiform convulsions. A peculiar oscillation of the eyes has also been noticed, but of this we shall have more to say later.

Choreic Movements have been reported in miners who have been exposed to after-damp. These movements may be so exaggerated as to simulate epileptiform convulsive movements. After the Courrières disaster, Dr Dervieux found that the development of choreic movements was by no means uncommon. Dr Shaw Lyttle also describes a case where a survivor of the Albion explosion was found in a very noisy condition, crying out loudly. Next day he was quite insensible. He continued for a few days in a peculiar mental condition, when he developed well-marked choreic movements which lasted for a few days. He was also affected for some time afterwards with double vision.

Sometimes the arms and legs are found in a *peculiar rigid condition* even where there have been no convulsions; an arm, for example, remaining quite rigid for a day. We have observed the same rigidity in cases of coal-gas poisoning. *Trismus* has also been described in survivors from mine explosions; the clenching of the teeth may continue for more than a day.

Convulsions.—Convulsions are not so common after exposure to after-damp as after furnace-gas, producer-gas, and illuminant-gas poisoning. Only two cases in connection with collieries have come directly under our notice. The first was a young man who was severely gassed by breathing the gaseous products of combustion of blasting powder in a place deficient in ventilation. He was unconscious for an hour, artificial respiration being resorted to in the pit. On becoming conscious he was driven home in the afternoon. At night he took three convulsions. He complained of severe pain in the pit of the stomach and of violent headache, was "heady," and shortly before the convulsions set in, vomited twice. After the convulsions passed off he regained consciousness and made a good recovery, although he was not able to resume work until a couple of months later. During that time his wife declared his temperament to

have become completely changed; from being a quiet, peaceable man he had become quarrelsome, suspicious, easily upset over trifles, readily excited, and forgetful. He also lost weight, and complained frequently of pain in the epigastrium, loss of appetite, and frequently-recurring headaches and giddiness. These symptoms, however, both mental and physical, soon passed off; he returned to his normal frame of mind, and made a perfect recovery. The other case was one which ended fatally. A young man was engaged building-off a waste-fire, when he was overcome by gas and was dragged out in a comatose condition. Convulsions supervened, and were the most marked feature of the case. He died thirty hours after the accident, without regaining consciousness.

Dr Shaw Lyttle[1] describes several cases of epileptiform convulsions among the survivors of the Albion explosion. In one case in which the pulse was found at first remarkably slow (38 per minute), six days later there were marked spasms of the face and also opisthotonos, the patient dying eleven days after the explosion. Survivors of explosions may be found in an extremely restless condition. The same observer has reported such a case in a man who was found tossing himself from side to side and striking his head on the ground.

Convulsions are much more common where the percentage of CO is high and the poisoning occurs rapidly, as in cases of poisoning by producer and illuminant-gas, and also where the poison acts in more confined places after blasting operations. For example, Dr Josephson[2], who had considerable experience of gassing by the fumes of blasting powder used in the demolition of the fortifications of St Juliers, frequently found that convulsions developed in the most serious cases. At the Crarae disaster[3] several of the survivors had more or less severe convulsions.

In what the French call "mal des mines"—poisoning by fumes from blasting in the galleries of war mines—convulsions in the victims have frequently been described by both French and German doctors. It was found that on dull days poisoning might take place even a considerable time after explosions, owing to the toxic gases having become infiltrated into the surrounding earth and crevices and fissures of the soil, and being later liberated. In one case where there were very severe convulsions, Rigal[4] observed contracted pupils and generalised anæsthesia of the skin, which lasted for some time.

[1] Shaw Lyttle: Haldane's Report, "Causes of Death in Colliery Explosions," etc., App. B, p. 38.

[2] Josephson: *Annales d'Hygiène Publique*, 1860.

[3] Crarae Disaster. Report by Col. Ford, 1886.

[4] Rigal. Quoted by Artigalas. *Des Asphyxies Toxiques.* Thèse de Paris, 1883, p. 33.

Cardio-Vascular Symptoms.

It has already been noted that the cardio-vascular symptoms, viz. :—palpitation, breathlessness, oppression about the chest and præcordial region, tachycardia, and throbbing of the large blood-vessels in the neck, are very often complained of in poisoning by after-damp. When these are aggravated by exertion, cerebral symptoms frequently supervene, such as vertigo, severe headache, ringing in the ears, flashes of light before the eyes, and, in some cases, faintness with a feeling of impending death. These symptoms usually pass off in a few days, but they may last much longer, returning, for example, whenever the patient over-exerts himself.

The following interesting case quite recently came under our notice. A man about sixty years of age and his son were poisoned by CO which found its way into their working place from a fire which had been built-off. Both were able, after recovering to some extent, to go home. On arriving home the father collapsed and became delirious, and continued in this state for four days. On the second day he started coughing, and at night a brisk hæmoptysis began which lasted three days. During the first week he had shivering fits and frequent attacks of faintness. He also complained from time to time of breathlessness, oppression about the chest, palpitation, and tachycardia. He had repeated attacks of giddiness and almost constant headache. These symptoms lasted three weeks. Then he attempted to go out-of-doors, but the exertion of walking brought back all these previous symptoms; he felt quite helpless and began to stagger; he declared that the upper part of his body felt as if it were squeezed by something. Two months afterwards the simple exertion of walking in the room and sharply turning round increased the heart's action by 20 beats per minute. No organic lesion could be made out in any of the organs. By the end of three months he was very much improved, and could walk a considerable distance without being troubled in any way. Then he had a relapse, and died suddenly.

In some cases the damage done to the heart and blood vessels may be permanent. It was undoubtedly the action of CO on the heart, while assisting in rescue operations at Snaefell Mine disaster, which ultimately led to the death of Sir Clement Le Neve Foster. Later, we report in full a case in which the damage done to the arteries was permanent. In producer-gas poisoning it is frequently on the heart that the brunt of the action of CO gas falls; and cases of sudden syncope frequently occur after poisoning by after-damp. Not infrequently, however, just when the signs and symptoms are pointing to the patient's early recovery, one may be disappointed to find the symptoms of cardiac weakness suddenly develop and death ensue from heart failure.

In poisoning by after-damp there is at first generally a high tension pulse of increased rate, 80 to 90 per minute, which may last for some

time. As the case gets worse the tension generally falls, and this is followed by a very rapid and weak pulse which may be hardly perceptible at the wrist. It may be irregular and intermittent. In some cases we have noticed very marked *tachycardia*, the pulse running at the rate of 160 per minute.

Tachycardia is due to paralysis of the pneumogastric or to stimulation of the sympathetic nerves or the intracardiac ganglia. In some cases the pulse may become remarkably slow; for example, in a case recorded by Dr Shaw Lyttle[1] it was only 38 per minute. A young man aged twenty-one was brought up from the Albion pit in an unconscious condition after the explosion on the morning of the 20th June; at night he recovered consciousness; next day he remained conscious but was very restless at night; on the 26th he remained nearly unconscious, with pulse only 38 per minute and easily compressed, the pulse remaining at that rate for three days; on the 28th he became partly conscious, with a pulse rate from 46 to 55; on the 29th the pulse was 88, the temperature 100·5° and he was again unconscious; on the 30th he was still unconscious. There were also spasms of the muscles of the face and limbs and opisthotonos. He continued in this condition, gradually, however, becoming weaker, bed sores formed, and he died eleven days after the explosion.

Bradycardia or decreased pulse-rate, on the other hand, may be due to stimulation of the pneumogastric or paralysis of the cardiac-sympathetic nerves and ganglia, and we know that poisonous substances circulating in the blood may cause marked slowing of the pulse by acting on the cardiac ganglia, etc. In uræmia, for example, where the various products of metabolism are circulating in the blood, the pulse may be very slow. Perhaps in CO poisoning, where, owing to the lessened supply of oxygen to the tissues through a large proportion of the hæmoglobin being fixed by the CO, we have incomplete metabolism and a consequent toxæmia: the same explanation may be offered regarding the remarkably slow pulse sometimes found in poisoning by after-damp, and by producer and illuminant gases. As a matter of fact it has been found during inhalation of CO in experiments on animals, that at one stage the pulse becomes very much decreased in frequency.

The high tension pulse may be explained in the same way, for this is found often in anæmias, owing to lessened and incomplete metabolism and the consequent circulation of waste products in the blood.

Certain vaso-motor disturbances are also met with, such as flushings of heat over the face, head, and neck, followed usually by cold sweats. Marked œdema, attacking either the arm or the leg, and curiously localised in most cases on the right side, has also been described by a few observers. The hands and feet may become quite dead to sensation,

[1] Shaw Lyttle: *Loc. cit.*, p. 38.

being physically white and cold in appearance. There may also be prickling sensations in the extremities. The numbness of the hands is well illustrated by the case of Sir C. Le. N. Foster, who, while engaged in rescue work at Snaefell, was overcome. He burned his hand and wrist with a candle, and had no idea that he had done so, till a friend in the evening called his attention to a large blister that had formed. Other examples of this as found in blast-furnace and illuminant-gas poisoning will be adduced. Patches of a reddish-brown character, raised above the level of the skin and insensible to pricking, have also been found, while it is a common occurrence to see patches of various sizes and forms of an erythematous character, sometimes of a cherry-red colour, distributed on the skin, especially over the chest.

Arterio-Sclerosis

One of the most unusual results of CO poisoning which has come under our observation was the development of what might almost be termed acute arterio-sclerosis in a man, the main facts of whose case are as follows :—G. A., aged forty-six, who was engaged "building-off" a waste-fire in Larkhall in May 1906, had previously been working for a long time in very bad air ; and had been employed at the above fire for nearly two days before he was overcome. The first day he worked at it for nearly two hours ; the next day for four hours. During that time, he and the men along with him complained very much of the effects of the gas, since they had to sit down frequently owing to breathlessness and weakness in the legs, and to hold their heads which ached very badly. While working they felt giddy and "quite silly on their legs." Next day he was engaged at the same work, and after a very short time four of the men were seriously affected and became unconscious. One of these died, and another, after being in convulsions for a day, died without regaining consciousness. G. A. worked hard to get the other men out. While thus engaged he felt very ill, had racking headache, giddiness, breathlessness, severe oppression in the chest, and experienced loss of control over the legs. He was taken home. Since that time till now (1914) he has not been able to work. On getting home he was in a most excitable state, and staggered about like a drunk man ; besides, he had a feeling of cold in his legs from the knees downwards, and in his hands and forearms, which became very white. The feeling of oppression in the præcordial region and in the chest, instead of disappearing in time, got worse, and he had attacks of severe palpitation and from time to time of violent headaches "as if his head were to burst open." With the headache there was accompanying severe vertigo. While these attacks lasted, he sometimes had the feeling of impending death. He felt well so long as he lay in bed, but whenever he made the least exertion these symptoms

appeared; indeed the taking of a full meal would precipitate an attack. Once he fainted and was unconscious for more than ten minutes. After an attack, when his doctor arrived, his pulse was found to be very slow. He has never had anything of the nature of a convulsion. His appetite and digestion had all along been good. Since the accident he has become much more excitable and nervous, and his memory very bad. His intelligence, however, was never of a high order. His hands and feet often become quite white and cold, as if dead. He has also been troubled with great flushings of heat.

As his condition got worse he was sent in 1906 to the Western Infirmary under the care of the late Dr Lindsay Steven. The following is a report of the cardio-vascular system as found in the Hospital Journal, all the other organs being found normal:—

"Pulse is regular but of very high tension. The vessel walls are like whipcord and can be pulled out. They are not calcareous. S.P. = 240 mm.; D.P. = 160 mm. A sphygmographic tracing shows a fairly high tension pulse with a high tidal wave. Apex beat is faintly palpable in fifth interspace and is easily felt when lying on side. Cardiac area is very slightly increased; cardiac hypertrophy, if it exists, is not marked. There is no marked accentuation of the second sound, but at the apex the first sound is found loud in tone, and slightly prolonged and booming in quality. No murmur could be made out. Knee jerks slightly exaggerated."

When we saw him in July 1908 ($2\frac{1}{4}$ years after the accident), we found him much improved, the oppression, etc., had disappeared, and he was now able to walk about without bringing on the distressing symptoms. His arteries were not nearly so hard nor was the pulse of such high tension; indeed they were fairly soft and compressible. Formerly he could actually take hold of his radial arteries and pull them out like elastic cords; this he could not do now. His arteries show no signs of atheromatous changes. His hands and feet no longer became "dead." His mental condition was not greatly improved, however, for his memory was still very bad and he was excitable and nervous. Indeed, when we saw him, his appearance was highly suggestive of a man in the first stages of intoxication, with flushed face, uncertain, jerky speech, and restless manner, although he had taken no liquor since the time of his accident. He has never had syphilis or renal disease, and plumbism, gout, rheumatism, and other toxic conditions may also be excluded. Before the accident he was in the best of health, not having known a day's illness.

In connection with his claim for compensation we had to examine him two years afterwards, in September 1910, when we found that his condition had changed for the worse. Even walking a very short dis-

tance brought on a feeling of oppression in his chest, breathlessness, and palpitation, his legs gave way, and he staggered and sometimes fell. The palpitation kept him from sleeping. He had attacks of complete collapse with blindness and a feeling of impending death. He complained of most severe headaches at times, and noises in his head "as if a band were playing." He had also attacks of vertigo with a "gone feeling," as if his head were going round and round. Just before these swimming attacks came on he felt the ground under him quite soft, and then he could not feel his legs. His arteries showed further degenerative changes, the pulse, which was small and frequent, being 118 per minute when he was sitting, while the exertion of walking in the room and turning quickly brought it up to 142. There were also changes in his heart, which was now enlarged, and the sounds were most suggestive of degenerative changes in the heart muscle. He was still excitable and nervous and his memory was very bad. The condition remained practically the same when we examined him in 1913.

With regard to the development of arterio-sclerosis, two explanations as to its production may be offered, both brought about by anæmia of the tissues. Before describing them, let it be remarked that Klebs held that the most characteristic action of CO was vascular dilatation owing to paralysis of the vascular walls, and that the arteries at certain points became elongated and tortuous, these conditions exercising pressure on and causing anæmia of the surrounding brain tissue. High blood pressure may in some cases of CO poisoning be actually of a compensatory nature produced by anæmia of a vital part of the brain, *e.g.* the medulla, the persistent high pressure leading to organic changes in the vessel walls. It is well known that if a vital part, such as the medulla, has been rendered anæmic by defective blood supply due to narrowing of the lumen of vessels by disease or by pressure of a new growth, the blood pressure may rise considerably in order to give the tissues sufficient blood at all hazards. The other and more probable explanation of arterio-sclerosis following CO poisoning is, that owing to the combination of the hæmoglobin with CO, there is oxygen-starvation of the tissues, the result of this being defective metabolism, with the consequent circulation of intermediary products of metabolism which give rise to a toxæmia.

Symptoms which point to a case going on to a Fatal Termination.

Patients may be found in a deeply comatose condition and may die without regaining consciousness. They may remain unconscious for days. Most of these cases die. Indeed, when a patient does not recover consciousness in a few hours, the case must be regarded as very dangerous, because if and when he does recover it is often with certain defects,

chiefly nervous in character, which may be permanent. Nevertheless no case ought to be regarded as hopeless, for some extraordinary cases are on record of patients who had been unconscious for days and yet who made a perfect recovery.

Some have been found in a condition not unlike the symptoms of cerebral hæmorrhage, *i.e.*, unconscious, the face flushed or pale, with profuse perspiration, and with stertorous breathing—some of these cases die without regaining consciousness. We do not generally see convulsions in cases of poisoning by after-damp, and the positions of the bodies as found rarely suggest that they have died in convulsions. However, a few have been discovered in epileptiform convulsions, and a number of these die. In building-off an underground fire in Larkhall, one man who was taken out in a comatose condition died after being a day in convulsions.

After large explosions when men develop convulsions they often die. Indeed, we should say that cases with convulsions which come on after an explosion, where the patient has been exposed for a long period to a small percentage of CO, and where there has been prolonged oxygen-starvation of the tissues, are very much more serious than cases where convulsions set in after exposure to gases with a high percentage of CO, such as producer-gas, where, although more red blood corpuscles are thrown out of action and consequently oxygen-starvation is greater at the time, the action of the gas is not so prolonged and therefore the damage done to the tissues is not so great. The same is the case with pneumonia; its onset after exposure to after-damp is to be dreaded, as it generally has a fatal termination. It is much more serious than pneumonia following poisoning by producer-gas and illuminant-gas, and for the same reason.

A very slow pulse is serious, as it points to paralysis of the cardiac sympathetic nerves and ganglia. Tachycardia also, if it persists for any length of time, must be regarded as serious, as it points generally to grave injury to the vagus nerves. In severe cases the pulse is generally very rapid and hardly perceptible.

In a few cases it has been noted that the *temperature has risen considerably before death*, and when the temperature is high during the illness the prognosis is grave. Dr Shaw Lyttle[1], for example, describes one such case in a survivor of the Albion disaster who was brought up unconscious, but had a relapse next day, when his temperature rose suddenly to 105°F. During the next two days he got gradually worse, with a temperature of about 104°, pulse 130, and epileptiform twitchings. Next day before he died there were clonic spasms of the limbs, and a pulse of 130 and very weak, the temperature being 104·5°F.

[1] Shaw Lyttle: *Loc. cit.*, p. 37.

Death in any case comes on very quietly. It is generally painless, the senses being in a manner gradually lulled to sleep before the end. In some cases the symptoms complained of at the time may be very slight, and yet in a few days very serious ones may develop.

Again, the patient may appear to be going on very well and may be able to be out-of-doors, when suddenly he has a relapse and dies. A notable and characteristic feature of poisoning by CO is that the symptoms come and go, the patient one hour being fairly well, and in a short time seriously ill, and this fluctuation from good to bad and from bad to good may occur several times in the day.

Sudden Death.

It is very important, then, to remember that even in slight cases of poisoning, where none of the signs and symptoms points to danger, the patient may develop serious sequelæ which may last permanently; and that cases of sudden death have also followed what appeared to be comparatively slight poisoning, and where everything seemed to point to a speedy recovery. In the Transvaal, Pakes[1] found "that a man may be badly gassed and apparently recover, and yet die as the result of the gassing. It does not follow at all that because a man has regained consciousness, and even the use of his limbs, he will therefore recover. Nor does it follow because he died many hours after, that he must have died of some other form of poisoning." We have found, in regard to giving evidence before Courts of Law, that it is difficult to persuade the Court that very slight cases of gas poisoning may lead to serious after-effects, and that a patient does not necessarily require to be rendered unconscious for dangerous sequelæ, and even death, to ensue. This point is not sufficiently appreciated by medical men unacquainted with CO poisoning although there is now abundant evidence of the fact.

Several deaths occurred among survivors a considerable time after the Courrières disaster[2] who were considered to have recovered from the CO poisoning. These took place very suddenly and unexpectedly. After a recent serious colliery disaster in New South Wales, it was found that sudden death occurred among a few who had all the appearance of having recovered from the primary attack.

W. H. Chambers[3] reports a case where a man died very suddenly after inhaling CO while engaged in a mine "digging out a fire." He suddenly complained to his mate of feeling faint and ill, and he was sent home. He lay on a sofa during the night. In the morning about six o'clock his mate asked him how he felt; he replied that he felt no

[1] W. C. C. Pakes: Transvaal Phthisis Commission Report, p. 139.

[2] Firmin Dervieux: *Loc. cit*, p. 540.

[3] W. H. Chambers. "Notes on Gob Fires." *Trans. Inst. M.E. Vol. XVIII.*

better, and almost immediately sighed and died. At the inquest, the doctor who attended him stated that death was the result of heart failure following CO poisoning.

A case occurred in Lanarkshire a few years ago where a young man, who had been working in a very badly-ventilated place, where there had been a good deal of shot-firing, was gassed. He was taken home and put to bed, where he died very suddenly. The case was heard before a Sheriff with a medical assessor, and although everything pointed to CO poisoning, the case was judged to be one of cerebral hæmorrhage. In connection with this decision, we should add that twenty or thirty years ago, owing to the finding of hæmorrhages in the brain following illuminant-gas poisoning, a not uncommon verdict was death from cerebral hæmorrhage, accelerated by inhalation of coal-gas. In the above-mentioned case we should add that no *post-mortem* examination of the cranial cavity was made, although the rest of the body was examined by a doctor. In several recent cases of sudden death following poisoning by producer-gas, the verdict returned by the Court has been death by syncope accelerated by carbon monoxide poisoning.

One of the most dramatic cases of sudden death is that given by Artigalas.[1] Two men, who were working at the bottom of a branch of a war mine, feeling themselves ill came out. The same thing happened with some other soldiers next day. On the following day a lieutenant went almost to the end of the gallery, and on coming up spoke a few words to the men under him, declared that there was no danger, and immediately fell dead. A *post-mortem* examination did not show any lesion to account for this sudden death. A German Commission, which inquired into cases of gassing in connection with war mines, mentions the fact that such cases do occur, but they did not specify any cases.

In most of these cases, death has in all probability been caused by the paralysing action of CO on the nerves of the heart or on the centres in the medulla. A few deaths may possibly be explained by the punctiform hæmorrhages in the brain which have been described by various authors.

[1] Artigalas: "Des Asphyxies Toxiques." *Thèse de Paris*, 1883, p. 36.

CHAPTER VII.

Laryngitis, Bronchitis, Etc.

In some cases severe inflammation of the upper air passages, such as pharyngitis, laryngitis, tracheitis, and bronchitis, are met with. These conditions are frequently encountered among the survivors of large colliery explosions and those exposed to the fumes from underground fires and burning charcoal, but are also seen after poisoning by producer, furnace, and illuminant gases. The inflammatory condition may be either of a simple catarrhal nature, or of such a severe character that necrosis of several parts of the mucous membrane results. Inflammatory conditions have also been observed in the mouth and nose.

In a case recorded by Ziemssen,[1] for example, of a woman of 40 years who died three days after CO poisoning, a diphtheritic exudation was found covering the hard and soft palates; and in a case by Thomson,[2] inflammation of the mucous membrane of nose, mouth, and throat was observed. A remarkable case has been reported by Putz[3] in which three children, the eldest of whom was five, were poisoned by charcoal vapour, and who developed a remarkable laryngeal condition. The larynx was covered with a croupous membrane which caused stenosis of the passage, necessitating tracheotomy. Through the wound casts covered by particles of coal-dust were removed. In one of these cases *post-mortem* examination showed that this croupous condition had passed down into the bronchi, and there was also well-marked lobular pneumonia.

Brouardel[4] mentions a case of a woman 28 years of age who developed acute laryngitis and bronchitis after exposure to the fumes from a fire. For a few days there was complete aphonia, and for four months the voice remained very hoarse. In this case the menses were suspended for two months.

Acute catarrhal bronchitis is frequently seen among men who return to their places too soon after the firing of shots in mines. In the Transvaal there have been many such cases, and in a certain percentage of these the cause was the irritating nitrous fumes. (See also p. 117).

[1] Ziemssen. Quoted by Sachs. *Die Kohlenoxydvergiftung, Braunschweig*, 1900.
[2] Thomson. Quoted by Putz.
[3] Putz. Inaug. Dissert. Halle, 1882, p. 48.
[4] Brouardel: *Loc. cit.*, p. 295.

Development of Asthma after Exposure to Fumes from Blasting

A miner, aged 23 years, was working in a place where there was a considerable percentage of black-damp, and as the place was very badly ventilated, the fumes from blasting operations were not quickly dissipated. He was warned by his companions not to go back to his place, but he ignored their warning, with the result that he collapsed unconscious on the pavement. He was dragged out into the main road by his companions, who had considerable difficulty in bringing him round. He went home complaining of a racking headache, giddiness, and ringing in the ears, and had also a severe attack of vomiting. An acute attack of bronchitis then set in, and during recovery from this he had an attack of asthma, from recurring attacks of which he suffered for the next six weeks. From this he recovered completely, and returned to his work after two and a half months' absence. He had enjoyed the best of health previous to being gassed, never before having had an attack of asthma or bronchitis, nor has he had any return of either since, and that is several years ago.

Pneumonia.

The question of the development of pneumonia in miners exposed to CO and to vitiated air (that is, air with a high percentage of CO_2 and a decreased percentage of oxygen), is one of the greatest possible importance from the fact that such cases are now and again presenting themselves in the Courts for compensation.

The following is a brief sketch of a test case in Lanarkshire in which both of us recently gave evidence. Two men, Robert and Patrick K., who had just fired a shot of about 1 lb. of gunpowder in their working-place, returned after an interval of only three minutes and while the place was still full of smoke. Both complained in a very short time of the state of the air. Their work appeared to them more difficult than usual, and they both suffered from headache and giddiness followed by vomiting. Owing to their condition they had to stop work four hours earlier than usual. At the pit bottom Robert was very ill. There they were kept waiting, exposed to cold and draught, for two and a half hours before being allowed up. Robert staggered home feeling ill, and complaining much of headache and giddiness. When his wife saw him he was staggering, and as his speech was curious, she could hardly make out what he said; in short he had the appearance of being drunk. He vomited a good deal that night. The next two days, although he still felt giddy and weak and was complaining of the racking headache, he went to his work. On the following night he developed lobar pneumonia,

from which he died on the fourth day. The Sheriff in his interlocutor held that "the illness which the deceased felt shortly after resuming work after the explosion (shot-firing) in his working-place on the 27th June 1910, and from which he was still suffering on the following days, was caused by the inhalation of carbon monoxide gas generated by the said explosion." The case was appealed to the Court of Session, and the Sheriff's finding was upheld.

The following is another example of the type of case which comes up in Court. This case came before the Sheriff and a jury at Dunfermline, and it was held that pneumonia following upon exposure to the fumes from firing heavy charges of gelignite was the cause of death. From the medical evidence it was also held that had a blanket and stimulant been available for the man when he came up from the pit, the pneumonia might have been averted.

We believe that it has been proved beyond doubt that pneumonia is not an uncommon cause of death in survivors from colliery explosions. After the Courrières disaster, for example, several men died from pneumonia. In these cases, described by Firmin Dervieux[1], the type of pneumonia was lobar and of an extensive character, the whole of the lung generally being affected. It was always the right lung which was attacked. Dervieux, in calling attention to this, a condition we may add which has been corroborated by other observers as well as by ourselves, also mentions a peculiar phenomenon which is very difficult of explanation, namely, that the different lesions which occur after CO poisoning, whether of vaso-motor disturbances, neuritis, paralysis, or pneumonia, are located by preference on the right side of the body.

In the Hulton disaster[2] (December 1910), in which 343 men perished, a few of the survivors developed pneumonia. One man who was badly gassed, developed pneumonia next day, the disease running the ordinary course and terminating on the tenth day. He was violently delirious for three days before the crisis. Another survivor was severely gassed, and only recovered consciousness on arrival in Bolton Infirmary. In the evening he was apparently so well that he was on the point of being sent home when he developed a severe attack of pneumonia from which, however, he recovered.

Regarding the type of pneumonia found, mention should be made of the case of one of the survivors of the Darran disaster. This was a boy aged 17 years, who was taken out unconscious and breathing stertorously, with parts of his hands, forearms, face, and neck superficially burned, in whom broncho-pneumonia developed five days afterwards and caused his death. Dervieux found in his case that there was a very high tem-

[1] Firmin Dervieux: *Etude Médico-légale de la Catastrophe de Courrières. Annales d'Hygiène Publique*, Nov. 1906.

[2] W. A. Hatton: "The Hulton Colliery Explosion," *B.M.J.*, 20th May 1911.

perature; but in cases which have been reported as following charcoal fumes poisoning and poisoning by illuminant and producer gas, one of the remarkable features frequently met with was the relatively low temperature as compared with the very rapid pulse rate.

Probably the most valuable evidence of the action of carbon monoxide in causing pneumonia has been furnished from the mines in South Africa and Australia. The results of the investigations of the Committee of Mine Medical Officers, which was appointed in 1903 to inquire into the causes of the high mortality among miners on the Rand, showed that the excessive death-rate was caused by a few diseases, chiefly respiratory, of which pneumonia was one of the most important. In the report issued by the Transvaal Miner's Phthisis Commission,[1] medical opinion was unanimous that "bad ventilation, air vitiated by gases produced by explosives, mis-fires, or otherwise, lower the power of resistance in the miner and tend, therefore, to render him more liable to pulmonary disease, miner's phthisis included"; and nearly all the witnesses, lay as well as medical, held that dust and gases were the commonest causes of respiratory diseases in the men who work underground. Some, for example, held in regard to the development of phthisis, that the gas first irritated the respiratory passages, then lowered the resisting powers and the nutrition of the lung tissue, and by doing so allowed the dust to do more harm than it would otherwise have done. Dr Irvine, in his evidence, said that he had seen a large amount of pneumonia and also meningitis due to the pneumococcus among underground workers; and Dr Aymard held that the breathing of bad gases played an important part in the causation of phthisis and pulmonary diseases, the gases weakening the tissues to a considerable extent.

Drs Irvine and Macaulay,[2] writing of the causes of the excessive death-rate, point out that "of these diseases, pneumonia was by far the most deadly," that in 1903 "the line of pneumonia dominated the whole mortality curve, and that during the latter half of 1903 no less than 63 per cent. of the total mortality was found to be attributable to pneumonia, phthisis, and other respiratory diseases." As one of the contributory causes of this, they were of opinion that the pollution of the underground air by dust and by noxious gases produced by explosives was most important.

The pneumococcus is a very virulent organism causing, as well as pneumonia, a very high mortality from cerebro-spinal meningitis. W. C. C. Pakes,[3] who gave evidence before the Commission respecting the effect of fumes in the causation of pulmonary diseases, laid the

[1] Report of the Transvaal Phthisis Commission. 1902-1903, p. 141.

[2] Irvine and Macaulay: "The Life History of the Native Mine Labourers in the Transvaal." *Journ. of Hygiene*, Vol. VI., 1908, p. 144.

[3] W. C. C. Pakes: Report Transvaal Phthisis Commission, 1902-1903, p. 141.

greatest stress on the action of CO in lowering the resisting powers of the lungs. He held that it was by far the most dangerous gas in the mines, and that compared with it nitrous fumes are almost negligible. With regard to this latter point the report says, "contrary to general opinion the proportion of nitrous fumes in normal mines is shown to be of small account. Possibly the undue prominence given to the presence and evil effects of nitrous fumes in the mines is due to the pungent odour and immediate irritating action on the mucous membrane which would tend to mask the existence of the more poisonous but less noticeable CO." Macaulay and Irvine, in the article already quoted, say: "it is possible that a certain proportion of the cases of extraordinary acute pneumonia which are seen amongst the natives may be due to the irritant poisoning by nitrous fumes." We think, however, that many of those who gave evidence before the Commission, more especially the lay witnesses, laid far too much emphasis on the effects of this agent. To begin with, in the many analyses of mine air which have been carried out immediately after blasting operations, and which we have previously detailed, there has been a surprisingly small percentage of nitrous gas present, and the characteristic pungent, irritating action of the fumes would prevent the bulk of the men going back to their places before these fumes had settled; moreover, it is found that they settle long before the more poisonous carbon monoxide gas has been dissipated.

Pakes[1] was strong on the point that in the Transvaal the air of the mines was exceedingly bad, and that the constant breathing of this would result in the men becoming anæmic and less resistant, more especially to respiratory diseases. He held further that the breathing of this bad air would have a very much worse effect on a man suffering from silicosis, and that as he had a part of his lung already thrown out of action for oxygenating purposes, his tissues would be supplied with less oxygen and it would be impossible for him to keep them in proper repair. Moreover, we may add, there would be imperfect metabolism, with formation of poisonous intermediary products and a resulting toxæmia. As such a man would have very much less hæmoglobin, he would be very much more likely to develop pneumonia. We have been told by "captains" or managers of mines in South Africa, and also by "shift bosses," that those suffering from silicosis stand gassing very badly; that pneumonia is frequently the terminal affection in such cases; that pneumonia frequently develops in the healthy native after he has been gassed, more particularly in fresh arrivals, who are regarded as more susceptible; and that the pneumonia which follows gas poisoning is generally fatal.

In Australia from 1880 there has also been a very great increase

[1] Transvaal Miners Phthisis Commission Report, p. 16.

in the death-rate from pneumonia. There, as in South Africa, it is put down to the action of CO from the too prodigal use of explosives, etc.

In view of this evidence, we hold that CO poisoning in mines is a very potent factor if not, indeed, the most potent in predisposing miners to pneumonia. In the two cases we have quoted, there was a difficulty in deciding how much the exposure for a considerable time to cold at the pit-bottom, after the body-heat had been already greatly diminished by the action of CO, had to do with the production of the pneumonia. It may be added, further, that in all cases of pneumonia following gassing in pits which come to Court for settlement, there is the additional difficulty that the true incubation period of pneumonia is imperfectly known, or is variable.

In some mining journals and books the inflammation of the lungs, which is distinctly recognised as a complication following explosions in mines, is put down to inhaling the hot air of the mine at the time of the explosion. If it were so, one would naturally expect that with such a cause death would be very speedy. But in all the Courrières cases, and in many others collected from mining journals, reports of explosions, and from other sources, there exists the history of the recent symptoms of CO poisoning, the giddiness, headache, feelings of oppression, tendency to sleep, staggering, and loss of power in limbs, etc.

Riembault [1] was the first to suggest that in large colliery explosions many of the deaths were not so much caused by asphyxiation from after-damp, as by the inhaling and swallowing of the red-hot particles of coal-dust suspended through the mines in an impalpable powder; that many of the victims were burned outside and inside by these particles; and that this was the actual cause of death. We have already shown from *post-mortem* examination of horses, etc., by Haldane and others that this is most improbable, and that most of the deaths are the result of CO poisoning. Dujol,[2] discussing Riembault's results of two *post-mortem* examinations where there were no traces of ulcers but a very vivid redness and swelling of the mucous membranes of the pharynx and larynx, believes that in explosions the gases of combustion inhaled at a high temperature produce nerve troubles; the nerve-endings of the pneumogastric in the pharynx, œsophagus, or lungs becoming the seat of a neuritis of an ascending character. We mention this in connection with pneumonia in mines, because a neuritis of the vagus (which has been demonstrated following CO poisoning) may so interfere with the nutrition of the lungs that they fall an easy prey to the pneumococcus; and also because we shall have occasion later to quote

[1] Riembault: *Comp. Rend. de l'Acad. des Sc.*, 15th April 1876. *Annales d'Hygiène Publique*, 1876, Vol. XLVI., p. 526.

[2] Dujol: "Une note sur les accidents morbides causés par les explosions limités dans les galeries des mines." *Bull. de l'Acad. de Méd.*, 1887, Vol. XVII., p. 175.

a case recorded by Mosso of pneumonia supervening in a man who was exposed to breathing very rarefied air, the cause of which he believed to be the result of paralysis of the vagus. For a number of years we have been inclined to the view that a neuritis of the vagus following the inspiration of the hot gases, etc., poured out at "slips" in furnaces, in which the men on the top are enveloped for a few seconds, might explain the death, and often sudden death, of a victim, where his burns were very slight and covered only a small area.

Cases of pneumonia have been reported following poisoning by black-damp and also by fire-damp; of the latter more particularly by German writers. In both of these circumstances we are dealing with a marked decrease in the amount of oxygen present, and in the first with an increase of the CO_2. The following is an interesting case in which pneumonia followed gassing in a man-hole. At Bermondsey,[1] London, a man descended a man-hole for the purpose of "coding" telephone cables, struck a match, which was immediately extinguished, and shortly afterwards he collapsed, but was rescued. A day or two later he developed pneumonia and died. Analysis of the air afterwards showed CO_2 2·5 per cent., Oxygen 15·10 per cent., and Nitrogen 82·40 per cent. There was no evidence of the presence of CO. It is probable that at the time of the accident the air contained much more CO_2 and considerably less oxygen. Indeed we should think the composition of the air was quite like the choke-damp of wells. However, the jury found that the deceased was accidentally poisoned by the inhalation of some gas, but that the evidence failed to disclose what that gas was; and that the pneumonia followed this gas poisoning.

Having carefully gone over all the clinical material at our command in literature, from our own personal experiences, from the experimental work of certain observers, and from the reports of many *post-mortem* examinations, we conclude that poisoning by charcoal fumes, and by furnace, producer, and illuminant gases is a marked factor in predisposing to the development of pneumonia.

One of the first cases in British literature in which pneumonia followed illuminant-gas poisoning was described by Sieveking[2] in 1869. Two grooms were overcome in a stable by gas which had escaped from a leak in a pipe, the fracture of which had been caused probably by a kick from one of the horses. Both were unconscious, but quickly recovered when oxygen was administered. The following day one of them complained of not being well, had pain and oppression in the chest, nausea, and headache. Shortly afterwards the symptoms pointed to pneumonia of the right lung. He recovered. Davison[3] also reports the case of a

[1] *Lancet*, 7th Nov. 1908, p. 1385.

[2] Sieveking: *Lancet*, 30th Jan. 1869, p. 159.

[3] Davison: "Case of Poisoning by Coal-gas." *B.M.J.*, 3rd Oct. 1896.

patient who died from pneumonia four days after being poisoned by coal-gas. Several cases are found in American literature following exposure to coal-gas which contained a very high percentage of water-gas. In one of these cases the patient developed double pneumonia two days after having been severely gassed, and died after an illness of two days. Firmin Dervieux also described a case of double pneumonia in one of the survivors of the Courrières disaster. One of us[1] mentions a case of a man and his wife who were poisoned by gas which contained 31 per cent. of water-gas, and consequently a very high percentage of CO, which escaped owing to defective connections in fitting up a gas stove. The husband, three days after exposure to the gas, developed congestion of the lungs with hæmoptysis. He brought an action for damages against the Gas Company, and was successful in proving that his illness was the direct result of CO poisoning. There are several curious cases of mysterious illnesses, afterwards proved to be due to coal-gas poisoning, which have been treated for a considerable time as typhoid fever or other illness, in which pulmonary symptoms were marked.

Many of the cases of pneumonia following CO poisoning occur after prolonged exposure to the gas, but it may develop after very short exposure, and also in cases where the poisoning was slight. Appolzer[2] mentions the case of a man of forty-two who developed pneumonia and hæmoptysis after slight poisoning by CO. The pneumonia here develops early after exposure, from thirty-six hours to three days generally. Bloch[3] reported the case of a young man seventeen years of age who was seriously poisoned by CO. On the second day there was an area of dulness at the base of the right lung and slight blood appeared in the sputum. This dulness increased next day, and was found also at the left base. He gradually got worse and died on the seventh day. Fraenkel's diplococcus was found present. On the last day the temperature, which had usually been between 38° and 39·5°C, rose to 40·5°C.

In many of the cases of pneumonia following CO poisoning, the temperature is found to be relatively low. Dufournier,[4] for example, reported a serious case of poisoning by the fumes from charcoal in an attempt to commit suicide, in which the whole of the right lung became consolidated with the usual signs of pneumonia, rusty sputum, etc. He was much struck by the fact that in this case the temperature only twice reached 100°F. We had a case of pneumonia following blast-furnace gas

[1] Glaister: "Water-gas, Carburetted Water-gas and Carbon Monoxide Poisoning." *Lancet*, 8th and 15th Dec. 1906.

[2] Appolzer. Quoted by Friedberg. *Vergiftung durch Kohlendunst.* Berlin, 1866, p. 122.

[3] Bloch: *Beiträge zur Kohlenoxydvergiftung.* Inaug. Dissert. Leipzig, 1902.

[4] Dufournier: "Pneumonie droite sans réaction febrile à la suite de l'intoxication par l'oxyde de carbone." *Gazette des Hôpitaux*, 1892, No. 89.

poisoning in a man who was engaged cleaning out a tube bearing furnace-gas to the steel work. The pneumonia set in thirty-six hours after the man was gassed. The whole of the right lung was solid, there was abundant expectoration of bright red blood, and, besides, severe abdominal symptoms, as meteorism, diarrhœa, and bladder trouble. The temperature was never above 100·5°F, but the pulse from the start was very rapid, and quickly became thready. The man, who was thirty-two years of age, and was regarded as a healthy and hardy worker, died within two days. A case following poisoning by suction-gas was reported from Worcester by one of the Factory Inspectors. The man was overcome by gas. On coming to himself, he staggered outside and lay down in the open air. When he had further recovered, he was taken home, and in a few days (the number is not reported) he developed pneumonia, but he recovered.

In a large number of cases in which coma has been profound, considerable œdema of the bases of the lungs may be found; as the day goes on this area of dulness extends, and there are signs of congestion of the lungs. These cases often die early, at the end of the first, or during the second day. Such a history undoubtedly indicates passive congestion owing to profound disturbance of the circulation.

Pneumonia may develop late in an illness caused by CO poisoning. This was seen in a case reported by Marthen.[1] A woman suffered for more than two months from various nervous disorders, disturbance of sensation, vaso-motor disorders, and an abscess over the sacrum. Then she had an attack of albuminuria, and a large area of dulness soon after developed over the right lung. No tubercle bacilli were found in the sputum at first, but later, acute and extensive tubercular mischief developed in both lungs, and she died at the end of three months after the accident. A case somewhat similar was reported by Bloch.[2] A boy of fifteen years was found in a ditch poisoned by CO from burning straw. When found he was unconscious, throwing himself from side to side; clonic spasms appeared in different muscle groups, *e.g.*, his thighs being drawn up on his abdomen. Bronchitis and acute pleurisy soon afterwards set in, followed by broncho-pneumonia and later by acute tuberculosis of the lungs, the patient dying at the end of two months. Children exposed to illuminant-gas have frequently been found to develop acute bronchitis or broncho-pneumonia. Lobar pneumonia is not so frequently met with in children as it is in adults. Rokitansky[3] reports a case where broncho-pneumonia of both lungs developed in a girl who died on the ninth day after poisoning by CO.

[1] Marthen: Beiträge zur Kenntniss der Kohlenoxydvergiftung. Virchow's *Archiv. Bd.* 136, p. 535, 1894.

[2] Bloch: Inaug. Dissert. Leipzig, 1902.

[3] Rokitansky: Poliomyelitis nach vergiftung mit Kohlendunst. *Wien. Med. Presse*, 1889, No. 52.

We have already drawn attention to a case where pleurisy was met with. Palma and Munzer[1] have reported an interesting case. The patient was found deeply comatose with the face cyanotic; but over the right zygomatic process a patch of white colour, two inches square, with slight infiltration of tissue underneath was found, a similar patch appearing on the right side of the forehead as large as half a split walnut. The skin over the thorax was reddish, and on the inner side of the right forearm there was a reddish swelling measuring three by two inches. Similar areas were found on both knees. Albumen, sugar, and a little acetone were present in the urine. The lungs were normal. Three days afterwards she developed pleurisy of the right side, with considerable effusion which quickly disappeared. In a case described by Klebs[2] there was also pleurisy with great effusion.

Pneumonia is not an infrequent terminal complication in CO poisoning. In a case recorded by Leudet[3], for example, neuritis developed on the eleventh day following gas poisoning. Twelve days afterwards the patient developed pulmonary congestion and died in two days. Fubringer,[4] Brouardel[5], and others have pointed out that after large fires some of those who escaped the immediate danger of the fire fell a prey to inflammatory œdema, purulent bronchitis, and pneumonia. Fubringer also found that in children a form of croupous laryngitis developed at the end of several days.

In the manufacture of nickel carbonyl one or two cases of poisoning have occurred, in which pathological changes in the brain and lungs have been described very similar to those found in CO poisoning. The lungs were found œdematous and intensely congested in one, and inflamed and solid as in pneumonia in another. These cases have been regarded as due to CO poisoning.

The following case[6] is interesting from the fact that although everything pointed to its being the result of CO poisoning, the doctor, under whose care the case was, held that this had nothing to do with its onset. On the 13th February 1905, a sailor aged forty-three, when intoxicated had gone into a Sailors' Home, turned on the gas, and went to bed. He was found unconscious and almost asphyxiated. Artificial respiration was performed for three hours in a draughty passage. On the 17th, when admitted into hospital, there was found "pleuritic friction in the left

[1] Munzer & Palma: Ueber den Stoffwechsel des Menschen bei Kohlendunst und Nitro benzol-vergiftung. *Zeits. f. Heilkunde,* 1894, p. 185.

[2] Klebs: Virchow's *Archiv. Bd.* 32, p. 503.

[3] Leudet: *Archives Générales de Méd.*, 1865, Vol. I., p. 513.

[4] Fubringer. Quoted by Vialettes. *Thèse de Paris,* 1895, p. 133.

[5] Brouardel: *Asphyxies par les Gaz.* Paris, 1896.

[6] Carter: "Some Old and New Remedies." *Lancet*, 5th May 1905, p. 1179.

axillary region, dulness of right pulmonary base, general congestion of both lungs, a swollen and acutely painful right shoulder blade, great distension of the abdomen, and a large slough on the anterior part of the tongue caused by forceps." He died the next day. At the post-mortem examination Carter looked for signs of CO poisoning, but found none. This was hardly to be expected after the lapse of five days. As he had also been informed by the gas engineer that no water-gas was supplied to the district, Carter concluded that the asphyxiation by gas had nothing to do with the causation of pneumonia. But did not the ordinary coal-gas to which he was exposed contain almost sufficient CO to kill the patient? This fact may have been overlooked. Oliver has found pneumonia developing in animals experimented on with CO; some of those which had not died at once of CO poisoning died a few days afterwards of pneumonia. On microscopic examination of the lungs small hæmorrhages into the air cells were found.

That pneumonia does follow CO poisoning is now recognised by a number of German writers, although some still support the old theory that where it is found it is due to the accidental insufflation of particles of food into the bronchi and lungs during vomiting. Vomiting, as has already been noted, is frequently found when the patient comes to himself after CO poisoning, and is one of the commonest after-effects; but pneumonia has appeared in cases of poisoning by after-damp, fumes of charcoal, and illuminant gases, where it could be clearly proved that there had been no vomiting. Klebs[1] described three fatal cases where considerable pneumonic areas were found in the lungs. He recognised that bronchitis, hæmoptysis, pleurisy, and pneumonia did follow poisoning by charcoal fumes and coal-gas, but he considered that these were accidental in some instances, and due to the insufflation of particles of food. Deglutition-pneumonia, he explained, was made possible by the anæsthetic action of CO at the beginning of coma. In other cases where pneumonia developed, he held that a lesion existed in the lungs prior to the poisoning. In one case quoted by him, the facts certainly pointed to the insufflation of food as the cause. In this case he found marked hepatization of the bases of the lungs, from which came a dull, brownish, foamy fluid in which yellowish crumbs of food were found. Becker[2] also had a case of deglutition-pneumonia, and was of opinion that a certain number of cases might arise in this manner. In some instances, he believed, the mucus of the mouth, with its teeming microbes, might be inhaled into the lungs which are already perhaps irritated by the gas, and the diminished sensibility of the bronchi would prevent its being coughed up before it reached the lungs and set up the inflammation.

[1] Klebs: Virchow's *Archiv*, Bd., 32.
[2] Becker: *Vierteljahrs. f. Ger. Med.*, 1893, pp. 113, 136.

Poelchen[1] also described a case where a man, twenty years of age, who was found unconscious in his room, developed after a few days deglutition-pneumonia. This became chronic. He coughed up a great deal of most offensive sputum. He developed pyo-pneumo-thorax, and died five weeks after the poisoning. One of Kleb's cases, which appears to us to be a straightforward case of pneumonia, occurred in a literary man who was poisoned by CO, and in whom two days later pneumonia developed in the middle and lower parts of the right lung, as well as an intensive inflammation and swelling of the mucous membrane of the mouth and throat and acute laryngitis. Friedberg[2] and a number of German authors consider that, although one might meet with a case of deglutition-pneumonia following poisoning by CO, the bulk of the cases were directly dependent upon the effects of the gas; that they were caused either directly by the gas acting as an irritant of the bronchi and lung tissue, or partly by the disordered circulation brought about by the CO. Liman[3] in his text-book says he has seen pneumonia frequently following CO poisoning.

We are of opinion, however, that although a few cases may be put down to the insufflation of food, the bulk of the cases are the result of the action of the CO. The *post-mortem* appearances of the lungs in nearly all these cases are against the view that pneumonia is commonly caused by insufflation of particles of food.

To complete the picture, we quote several cases of pneumonia occurring in persons breathing very rarefied atmospheres.

The theory, that the production of pneumonia in CO poisoning is aided by paralysis of the vagus nerve, is interesting when compared with Mosso's view that pneumonia in high altitudes is also the result of paralysis of the vagus. We have several times already drawn attention to the similarity between symptoms produced by breathing an atmosphere deficient in oxygen, as black-damp is, and those produced by breathing rarefied air. It will be well, therefore, if we consider some cases of pneumonia which have occurred at high altitudes. Mosso[4] describes in detail the case of a strong young man who developed pneumonia while mountain-climbing in the Alps at a very high altitude. One morning this man complained of shivering, general malaise, headache, sickness with vomiting, and depression, *his face being very livid.* At night he became *feverish*, and the case was diagnosed as one of pneumonia, the base of the *right lung* being affected. During his short illness the type of breathing with periodic pauses, which is so

[1] Poelchen: *Berlin. klin. Wochenschr.*, 1882., No. 26, p. 396.

[2] Friedberg: *Die Vergiftung durch Kohlendunst.* Berlin, 1866, p. 115.

[3] Liman: *Handbuch der Gericht. Med.* Berlin, 1889, Vol. II., p. 579.

[4] Mosso: *Life of Man in the High Alps*, 1898, p. 319.

commonly found in very high altitudes, and which is described by Mosso in detail, was quite marked. The temperature, *which was never high, fell by lysis.* The illness only lasted two or three days, and during convalescence symptoms appeared which pointed to there having been a small gangrene of the lung.

In considering how the pneumonia could have been caused, Mosso held that the probability of chill as a cause was not likely, because, if that had been the case, inflammation of the lungs would be very much more common in mountain-climbers, and this disease is much less frequent in the highlands than the lowlands, as indeed are all diseases of a microbic character. He also pointed to the anomalous course of the illness and the symptoms observed, to show that the cause could only be regarded as the result of breathing very rarefied air. In short, he regarded the case as *inflammation of the lungs brought about by paralysis of the vagus nerve.* In coming to this conclusion, Mosso was guided by the sudden onset of the illness and the rapidity with which the patient's condition became worse, for in six hours his condition was alarming, which is most unusual in pneumonia. The rapidity with which serious cardiac symptoms set in — almost at once the pulse became very rapid and barely perceptible, being countable at the wrist only with great difficulty—and the fact that this rapidity did not coincide with the temperature taken in the rectum, 37·8°C, were unusual. The other features of the case commented on by Mosso were, "the serious general depression, cyanosis, vomiting, the increased area of cardiac dulness, the extreme weakness of the cardiac impulse against the ribs, the irregularity in the rate and amplitude of the respiratory movements after the lapse of five hours, the pulmonary gangrene due probably to incomplete deglutition," all of which characteristic phenomena, said he, "can only be explained by paralysis of the vagus."

Another case recorded by Dr Jacollet[1] died of paralysis of the heart and lungs on Mont Blanc. Many of the symptoms were similar to those of the above case. The patient had no sleep the first night owing to constant coughing; next day he had a violent shivering fit, delirium, superficial breathing, temperature 38·3°C., and a pulse rate of 120. A *post-mortem* examination showed that he died of broncho-pneumonia. The meninges were markedly congested, and the vessels of the pia mater were enlarged and engorged with blood. Both lungs were congested and œdematous. Burdon Sanderson[2] also relates the case of a well-known German professor who developed pneumonia when ascending a very high mountain. He was brought down by his guide, and after a short but sharp illness recovered.

[1] Jacollet. Quoted by Mosso: *Life of Man in the High Alps.*

[2] Burdon Sanderson. Quoted by Mosso: *Life of Man in the High Alps.*

These observations of Mosso are most valuable, for may we not find in them, knowing as we do that nerve lesions are particularly prone to occur after CO poisoning in which there is a marked oxygen-starvation of the tissues, one explanation of some of the cases of pneumonia which follow exposure to this gas? From what has gone before, one can readily understand that pneumonia is more likely to follow long exposure to CO gas, it may be to small quantities, rather than in cases of fulminant poisoning where exposure has only been for a few minutes. This may explain the reason why, comparatively speaking, so many cases occur after explosions in mines where the survivors have been exposed for long periods to percentages of CO not large enough to cause death, but yet sufficiently great to throw out of work a large proportion of the oxygen-bearing red blood corpuscles, and therefore to lead to prolonged oxygen-starvation of the lung and nerve tissues.

From the evidence before us, we can point to certain peculiar distinguishing features in pneumonia succeeding CO poisoning. The cases generally develop rapidly after exposure, from one to three days—usually thirty-six hours; they are generally lobar, of a particularly extensive type; and very frequently the right lung is affected. The cases generally run a rapid course, patients frequently dying after an illness of two or three days' duration, sometimes even less, and a brisk hæmoptysis is often found ushering in the disease. In the majority of cases the temperature is low, *e.g.* in our own case, where everything pointed to an attack of a particularly virulent type, the temperature was never above 100·5°F. The pulse rate is out of all proportion to the temperature, becoming very early rapid and thready; the heart is quickly dilated; cyanosis appears early, and the apex beat can with difficulty be made out. In some cases there is marked vomiting, giddiness, and headache at the commencement, and in others, as in our own case, abdominal symptoms are prominent, diarrhœa and meteorism being present.

But when we come to consider the *relation of chronic poisoning by* CO to the causation of pneumonia we are face to face with great difficulty. This difficulty was quickly recognised by the Medical Inspector of Factories when he inquired into the prevalence of paralysis and pneumonia in an ironworks in the North of England. A medical man in attendance on the workmen informed the Medical Inspector that he recognised four types of illness among the workers :—(1) slight ordinary gassing due to CO; (2) severe gassing with headache and gastric disturbances causing absence from work for three or four days; (3) paralysis of a general paralytic type, of which there had been three cases in the last four years; (4) pneumonia of the Middlesborough type. He did not think, however, that the pneumonia was much more prevalent

among the workers at the smelting works than it was elsewhere. He had twelve cases, only one of which died. Another practitioner in the same district declared that he had had six cases of pneumonia all of which died but one.

When we consider gas poisoning in mines we find the same difficulty. It must be remembered in considering this question, that in chronic poisoning by CO the person has a considerable portion of his oxygen-carrying corpuscles put out of action during the whole of the time spent in the polluted place, and for a time also after coming out into the open air. The amount of oxygen which found its way to the tissues would therefore be reduced, and the resisting powers of the man, owing to this oxygen-starvation, greatly lessened. Pakes is very insistent on this point, that prolonged breathing of impure air gives rise to respiratory diseases.

The typical cases of lobar pneumonia which follow poisoning by producer or illuminant-gas, after-damp, or gases from blasting (either dynamite or gunpowder), and which come on from thirty-six hours to four days after the gassing, are due to the pneumococcus.

Pneumonia following Poisoning by Nitrous Fumes.

We have already pointed out that analyses of mine air, sampled immediately after blasting, have proved that far too much importance has hitherto been attached to the action of nitrous fumes produced by dynamite, gelignite, etc. *A small percentage of the cases of pneumonia are, however, undoubtedly initiated by nitrous fumes.* These we think are at first non-microbic, and are directly the result of the irritating action of the fumes on the lungs, causing intense congestion, with hæmorrhages and œdema of the pulmonary tissue. In these cases the period of incubation is not nearly so long as in lobar pneumonia after CO poisoning. In a few hours we find the preliminary irritative symptoms merge into most urgent symptoms, and within six hours of being gassed the patient has violent inflammation of the lungs which has generally a speedy and fatal termination. In other cases there may be few symptoms for three or four hours; then follows an illness of marked severity; and death may result within twelve hours. (Macaulay).

When a miner has inhaled a considerable volume of nitrous fumes, the first symptoms complained of are a feeling of burning in the nostrils and throat, with irritating dryness of the air-passages and a dry hacking cough; but these symptoms, however, quickly pass off. In a case described by Dr Wotherspoon of broncho-pneumonia following inhalation of nitrous fumes some hours after gassing, during which period little complaint was made, the patient was suddenly seized with

violent fits of coughing accompanied by great pain in front of the chest and in the left side. There was copious expectoration streaked with blood, the temperature being 102·5°F. Examination of the lungs showed a considerable area of dulness on the left side, and capillary bronchitis on the right side. In another case, after a quiet interval of an hour or less, violent coughing set in, with great difficulty in breathing, and a tightness at the lower part of the throat. Death resulted in twelve hours. In this case there was found intense congestion of the windpipe and bronchial tubes and acute broncho-pneumonia. The mouth, nostrils, trachea, bronchial tubes, and lungs were filled with froth. The lungs were injected in places, and there was also acute hæmorrhagic œdema.

Milder cases of poisoning by nitrous fumes are frequently met with. Drs Irvine and Watt have noted many of these cases, where the symptoms above described existed in a modified form, a common picture being "a quasi-asthmatic condition which lasts perhaps for twelve or twenty four hours or longer, with, perhaps, a little expectoration of blood-stained sputum."[1]

In most cases of pneumonia following CO poisoning, we have probably to deal with lowered resisting powers of the bronchial and pulmonary tissues, owing to the action of CO on the red blood corpuscles. Many of these are thrown out of action, with the result that we have oxygen-starvation of the tissues, which then become less resistant, so that the pneumococcus is able to make a successful invasion. If moreover, we are to regard chill as a predisposing agent, we have the characteristic action of carbon monoxide in causing marked loss of heat, or perhaps it would be more correct to say, in causing by its action on the heat-producing centres a diminution in the production of body-heat.

With the increase of clinical material, however, it is to be hoped and expected that this most important question will in time be more thoroughly and scientifically determined.

Neuritis and Paralysis.

Neuritis is another sequela of CO poisoning which has been described by those who have looked after the survivors of explosions. The facial, sciatic, and musculo-spiral nerves are among those most commonly affected. With regard to this, Dervieux[2] describes a very remarkable case which he deemed to be *neuritis of the phrenic nerve*, in one of the survivors of the Courrières disaster. This man when brought up showed the usual symptoms of CO poisoning, viz. :—headache, giddiness, drowsiness, etc. He suddenly developed œdema of the whole of the

[1] L. G. Irvine and A. H. Watt: "Miners' Phthisis" *Transvaal Med. Journ.* Sept. 1912.

[2] Firmin Dervieux. *Annals d'Hygiène Publique.* 1906, p. 538.

right arm from the fingers to the shoulder. The swelling was painless, white, hard, and not easily pitted. It gradually grew worse till he died. At the same time there was partial paralysis of the limb, the extensor muscles being principally affected; in fact, the paralysis was similar to that met with in lead poisoning. There was also complete anæsthesia in the limb. When Dervieux first saw him, the patient was quite sensible, his only complaint being slight hiccough. On examination the only symptom discovered was pain along the course of the phrenic nerve. The hiccough gradually became more troublesome, recurring at regular, short, periodic intervals. Everything was tried to stop it, but failed, and the hiccough went on without ceasing till his death which occurred a few days later. A little time before he died, signs of pneumonia in the right lung were discovered. The hiccough, it may be added, was present at least two days before the pneumonia, so could not possibly have been caused by it.

Partial loss of power in the limbs, more especially in the lower, has already been noted, but in some cases all the limbs are affected. Brockmann[1] describes such a case in a miner who was poisoned by after-damp from an explosion and thereby rendered unconscious. He was probably brought round by water dropping from the roof on his face, but he then found that he was quite unable to move owing to paralysis of all his limbs. The arms were the first to recover power, but more than two years elapsed before he could walk with any degree of freedom. Paraplegia is, however, much more frequently met with, and cases of monoplegia have been observed, an arm or a leg being involved.

Rare cases of hemiplegia have been recorded. Rigal[2], for example, reports an interesting case of hemiplegia of the right side which developed in a soldier who was working in one of the war mines. He was at first rendered unconscious. The surface of the body was quite cold. There was spasm of the glottis which interfered considerably with the respiration, and severe clonic and tonic convulsions developed. There was marked hyperæsthesia of the skin, the least contact producing convulsions. The day after the convulsions ceased, persistent somnolence developed and sensibility was abolished. On the fourth day intelligence returned, and it was found that the patient was suffering from right hemiplegia without implication of the face. There was absolute anæsthesia over the whole extent of the paralysed side; and there were areas also of anæsthesia on the left arm and over the pelvis, with diminished sensation over the chest and abdomen of the left side. The next day large congested patches were found on the chest, which disappeared, however, in the course of two days. On the ninth day

[1] Brockmann. Report of Prussian Fire-damp Commission.

[2] Rigal: Quoted by Artigalas. *Loc. cit.*, p. 34.

sensation returned to the left side; on the twelfth day he rose and attempted to walk, but dragged his right leg; on the thirty-first day after the accident, walking was still difficult; but by the forty-sixth day recovery was complete.

Paralysis may be met with in groups of muscles, as, for example, in the extensor muscles of the forearm and of the lower limb, causing wrist-drop and foot-drop. More uncommonly it is found that the paralysis is limited to one muscle. Artigalas[1] reports the following interesting case of a soldier who was overcome by blasting fumes. He had violent headache and vomiting, and soon afterwards developed convulsions; but in a few hours he was much better. Next day he experienced excessive pain in his left arm, which he found he could not raise. The deltoid was found to be completely paralysed, the muscle showing signs of degeneration in thirty-eight hours. The day after he was gassed, a red spot on the skin with considerable induration was found on the posterior border of the axilla. Artigalas believed that there was a neuritis of the posterior axillary nerve produced by the toxic gas.

These paralyses are most frequently exhibited on the right side, a phenomenon which several observers have pointed out, but which was specially noted by Dervieux in survivors from the Courrières disaster.

We shall be able to study these conditions more in detail under producer and illuminant-gas poisoning, as in these the clinical material at our command is much more extensive.

Nervous and Mental Symptoms.

The symptoms indicative of derangement of the nervous system brought on by CO poisoning in collieries are characteristic, and are so frequently found that one is forced to the conclusion that the action of CO is most marked on nerve tissue. Let us first of all in a general way review the mental and nervous signs and symptoms commonly exhibited.

To begin with, in certain cases the slightest anxiety or excitement will bring on a return of the symptoms primarily complained of; such as tightness and oppression about the chest, cardiac palpitation, and various pains and feelings of distress about the head, while beads of perspiration may appear on the forehead. In some instances the slightest mental or physical exertion will bring on these symptoms. It has also been found that excitable and over-anxious members of rescue parties are generally the first to feel the effects of the noxious gases.

It has already been said that when men regain consciousness, they appear dazed and stupid and generally have no recollection of what has happened. Their minds are confused, they seem to have no ability to

[1] Artigalas: *Thèse de Paris*, p. 36, 1883.

think, and they are unable to answer questions properly. Indeed, some of them look as if they were recovering from a drinking bout. Cerebral symptoms and changes in the mental state of the patient are recognised by army doctors as quite common in soldiers who are working in the galleries of war mines. Some writers, indeed, refer to the mental condition as "l'enivrement par la poudre," and also to the abnormal excitability and unusual irritability which develop in soldiers working in these mines. Artigalas[1] says that one of the most curious, as well as almost constant, of the symptoms is the unconscious laugh with a mimicking expression produced by the contraction of the zygomatics. This peculiar laugh is well known by such soldiers, and is regarded by them as a danger signal pointing to serious pollution of the atmosphere.

The patient at first may be constantly drowsy and sleepy, dropping off to sleep at all times and in all positions. At the same time he is indifferent, not troubling himself about anything; but later, an irritable phase frequently develops in which, instead of being drowsy, he is intensely wide awake and complains greatly of insomnia, while his mental condition is the reverse of apathetic, being most restless, excitable, and irritable. One medical man informed us of a case in which a man, after being gassed, changed from being anxious, hard-working, and energetic to being indifferent, easy-going, and contented, not bothering himself much about anything; and this mental condition lasted a considerable time. Depression of spirits, even melancholia, is frequently seen. Leigh[2] reports an accident where two men were gassed in a pit. One died, the *post-mortem* examination revealing cerebral hæmorrhage. The other, four months after the accident, was still unfit for work. He suffered much mentally, was extremely depressed, had lost his memory, suffered a great deal from insomnia, and ultimately fell into a condition of melancholia.

Another point to be noted is, that there may be failure to concentrate the attention on anything for any length of time, the result being that the judgment of the patient is apt to become impaired and he himself liable to arrive at hasty, ill-considered conclusions.

The ability for hard work, and especially the capacity for any operation requiring initiative are also lost. The patient is easily fatigued, the slightest mental or physical exertion almost prostrating him. There is generally loss of the higher cerebral functions; indeed, the patient may become for a time, what the Lanarkshire miner calls "weanly," that is, childish. He often becomes self-centred and, when so, is low-spirited and despondent. When this happens, we have at the same time

[1] Artigalas: *Thèse de Paris*, 1883, p. 37.

[2] Leigh: Report of Departmental Committee on Compensation for Industrial Diseases, 1907, p. 315.

generally found that he continues to complain of those symptoms which predominated at the time of the accident; *e.g.* oppression about the chest, headache, or some other symptom. In one case a man, hitherto most intelligent, complained to us that he had no proper control over his thoughts, in respect that these ran riot. In other cases there may be loss of stability of mind, of mental control, and of force of character. For a time, indeed, the brain capacity seems to drop to a lower level. In some of the cases the patient feels something wrong, but its nature he can hardly define or explain. He feels angry with himself, too, for he finds himself living as it were under a continual strain, without being able to explain it to himself or understand why it should be so, and all this further exhausts and irritates him. Such psychical phenomena as these should be studied along with those found after furnace, producer, and illuminant-gas poisoning, and especially in connexion with those cerebral lesions which are found on *post-mortem* examination.

Loss of Memory.

A very important derangement of mind met with is *loss of memory.* This loss is not only of recollection of what happened at the time of the accident,—a comparatively common complaint,—but also of previous events; in fact, a whole period of time may in some cases be erased from the mind. At the inquest on the deaths of thirty-four miners in the recent Hulton pit disaster, for example, a young lad, one of the two men in the pit on the 21st December 1910 who escaped death, was called to give evidence. He was found by a rescue party lying unconscious in a roadway. He was taken to the Infirmary and recovered. He was unable to throw any light on the disaster, however, as his mind was a perfect blank on that matter.

Bloch[1] reported a case where loss of memory, without any other disorder, was a prominent symptom following poisoning by the fumes from an underground fire. A miner had been sent into a place where gas had been escaping from a built-off fire. He was overcome and seriously affected, being unconscious for a few hours. On being taken to hospital he resisted and attempted to escape, all the time making inarticulate sounds. Next day he was much quieter, could be roused by shouting, but did not know where he was. He complained much of headache. The following day he was greatly improved and recognised where he was. He returned to work, but found that he was still troubled by the defect of memory. He would ask the same question again and again, and forgot his duties unless he wrote down what he had to do. This

[1] E Bloch: "Ein zür Heilung gekommener Fall von Kohlenoxydvergiftung mit Ausschbesslick psychischen Störungen." *Fortschr. der Med. Bd.* XX., 1902, p. 525.

interfered so much with his work that he had to give it up and go off for a month's holiday. On returning, the mental disorder had almost completely disappeared. For a considerable period, however, he experienced difficulty in learning anything by memory, or when he tried to count figures.

Minor cases of loss of memory may often be seen. Wives of miners, for example, will frequently volunteer information that their husbands when they are working in bad air, that is where the ventilation is bad and fumes from shot-firing consequently collect, are irritable, suffer from frequent headaches, are very drowsy and fall asleep when they get home, are roused with difficulty, and that they are very *forgetful.*

Dr Llewellyn[1] reports the case of a boy aged sixteen in the Albion explosion, who was brought up from underground (24th June) quite comatose, and who remained in that condition for thirty hours. He was occasionally delirious. He was able to go outside on the 26th June, but that night he again became comatose and remained in that condition till the 28th, when he appeared like a person *recovering from opium poisoning.* He was rousable but looked stupid, although he appeared to know what was said to him. He was very drowsy and in a state of melancholic stupor. He had no recollection of anything that had taken place. He gradually improved; but weeks afterwards he had still no recollection of what had happened. In the Courrières disaster one of the survivors spent twenty-four days in the pit after the explosion. Most of his companions had fallen by the way, and he had wandered about the main roads in an aimless fashion, subsisting on the food found on his dead companions. When discovered, he was found in a dazed, confused state, had no idea how long he had been in the pit, and, in fact, did not at all seem to realise his position.

An interesting point brought out by Dr Llewellyn is, that a patient when under the influence of CO has lost the sense of distance. One man, for example, thought he had travelled twenty or thirty yards whereas he had only gone a few feet. As that observer further remarks, the time-sense is also delayed; "every step is like ten." Sir C. Le Neve Foster, however, in relating his experiences at the Snaefell disaster says, that some of the members of the rescue party, though not absolutely unconscious, did not recognise the lapse of time, for they thought that ten minutes had elapsed between certain events while in reality it was two hours.

Cases with Pronounced Mental Symptoms.

As the following case offers a typical picture of the distressing action of CO on the mind, we shall describe it with some minuteness. It is

[1] Llewellyn: "The Darran Explosion from a Medical Standpoint." *Brit. Med. J.*, 11th June, 1910.

that of a man who, although he is now only fifty-six years of age, has already the appearance of an old frail man. He was one of those gassed while engaged building-off an underground fire. He was a contractor at the time of the accident, and as he was looked upon as one of the most capable and level-headed of the men, was told off to do what is correctly regarded as most dangerous work, viz. :—the "sealing-off" of a waste-fire. For three days he worked in a very poisonous atmosphere, and during that time had constantly to return to the main road for air. But one day he and five others who were working under him were suddenly overcome by the gas; two of these cases ended fatally. He himself felt sleepy, then gradually lost power over his limbs, and sank to the ground in a dazed, stupefied condition. He was taken out in an unconscious state. After regaining consciousness, he suffered from a swimming sensation in the head, loss of power over his limbs, and felt "everything going black." Curiously enough, this man did not complain of the intense headache which is almost invariably found in such cases. He had pain and oppression in the chest and palpitation of the heart with shortness of breath. The slightest excitement would cause these symptoms to become exaggerated, while even the slightest mental or physical exertion would bring them on. They were often accompanied by beads of perspiration breaking out on the forehead and face. He was now very nervous, easily excited, easily upset. He became depressed, indeed melancholic at times, and lost interest in everything.

Soon after the accident his hair began to get grey, and three years afterwards became quite white. He was not able to resume work until six months after being gassed. During that time he lived very quietly, and by medical advice passed his whole time in the open air taking long drives, as for a considerable period he felt that he had not sufficient control over his limbs to permit him walking any distance. Over-fatigue, even by walking short distances, would make him stagger. He suffered much from loss of appetite, and from being a stout and well-nourished man he beeame very thin and weak. For the first two or three weeks he was very drowsy and continually sleeping, but after that time sleeplessness was one of the things of which he complained most bitterly. For many years before his accident he was a large contractor having constantly a large number of men under him, among whom accidents with occasionally fatal results were not uncommon. We mention this lest it might be supposed that the symptoms described were those of neurasthenia, the result of shock to his nervous system occasioned by the accident and the loss of his two men. He was regarded as a very cool and resourceful man, and was generally picked out for work in any place where there was danger.

His present condition three years after the accident is interesting.

He looks like an old man, and has not the strength and vitality one would look for in a man of fifty-six years. His organs are perfectly sound and normal, but he finds that the least thing now depresses him and puts him out. Things which formerly gave him no concern, now appear of the greatest importance and moment. He cannot follow anything involved, and is not nearly so clear and logical in mind as he formerly was. There is also a loss of initiative, and he now feels that he could not possibly go back to his old employment of contracting, as he has not the mental power, grasp of detail, force of character and ability necessary to carry it on. He tried to force himself to fix his attention on some of the many little details connected with such work, but it required such painful effort that he could not persevere in it, for he felt very fatigued and quite ill after the mental exertion. Formerly, he could go almost anywhere, and the most dangerous work would not give him the slightest anxiety; but now he is quite timid, and has not the slightest confidence in himself.

In another case, a man was gassed while engaged in dealing with a "gob-fire," and was rendered unconscious for two or three hours, great difficulty being experienced in bringing him round. On recovering consciousness, he complained of breathlessness and palpitation of the heart, accompanied by severe epigastric pain which was relieved by vomiting. He had no control over his head, which rolled about in a helpless way when he was moved. His legs were affected, and he staggered when he tried to walk; his grip also was weakened. He was very drowsy, and was with difficulty aroused. For days he remained in a drowsy, dreamy state, very different from his usual energetic, alert manner. Then he developed another mental phase, becoming anxious, excitable, and restless, and sleeping very badly at night. His memory for a long time remained imperfect, although he had quite a good knowledge of all the events immediately before his accident. As in the previous case, so in this, there was a loss of power of concentration of attention on anything.

In another case in which the gassing was produced in a similar manner, the man was off work for four months. The symptoms of which he complained were very similar to those given above, being principally nervous. A strong, healthy man who had never been troubled with "nerves," and who was cool and not easily put out, was rendered unconscious with CO, and developed into a neurotic, timorous, excitable, unstable individual. This condition lasted for a few months after he commenced work, and even now, two years after he met with his accident, he is not the man he was, being very easily worried, upset, and excited over matters which formerly would not have given him a moment's consideration.

Disturbances of Speech.

After explosions it has been found that in some of the survivors speech is affected. In some instances speech is entirely lost, and only comes back after many days. In others there is great difficulty in forming the words, and as the patient's mind generally is slow and confused, there is a curious impression given to the observer of great effort in the attempts to speak. The patient often repeats himself, iterating and re-iterating words or phrases. This affection of speech may and generally does last only for a few days, but in rarer cases it may be months before speech returns to its normal: when this is the case there are generally, however, other mental symptoms.

Sir Clement Le Neve Foster's account of his personal experiences at Snaefell.

In his report on the Snaefell disaster, Sir C. Le Neve Foster added a most interesting account of his personal experiences of the effects of CO while he was engaged on that occasion in rescue work, the effects of which, it may be conjectured, undoubtedly contributed to his early death. As the mental phenomena described are of the most interesting and instructive character, we shall describe them at some length. When he and the other members of the rescue party were overcome by the gas, they were only a few feet from safety; and although they knew that fact, they could not muster the necessary muscular power to place themselves in safety. When he found himself being overcome, he took out his note-book and therein wrote a farewell letter to his wife and children.

In the report he draws attention to the repetition of certain words, and even of whole sentences, in what he had written. He remarks:—"I had absorbed enough of the poison to paralyse me to a certain extent and dull my feelings, but at the same time my reason had not left me. The general sensation was like a bad dream, and yet I was able to reason properly and write intelligibly though in a disjointed fashion. The following is the letter as written:—'2 p.m., goodbye, we are all dying, your Clement. I fear we are dying, goodbye, all my darlings all, no help coming, good-bye, good-bye we are dying, no help comes, good-bye, good-bye.'" Then further on, showing that he was still able to reason, he wrote:—"We saw body at 130 and then all became affected by bad air, we have got to 115 and can go no further, the box does not come in spite of our ringing for help. It does not come, it does not come"; then still further on:—"I feel as if I were dreaming, no real pain, good-bye, good-bye, I feel as if I were sleeping;" then later:—"2.15 p.m. men are all done, No . . .[1] or scarcely any, we are done, we are done,

[1] Here the word was illegible.

godo-bye (sic) my darlings ;" then again, " No pain, it is merely like a dream, no pain ; no pain, for the benefit of others I say no pain at all, no pain, no pain ! " [1]

When he was rescued and brought to the surface; he had a feeling of exhilaration and was in full possession of his senses, for he asked Dr Millar to take a sample of his blood. An hour afterwards he wired to his wife as follows :—" Am perfectly right, do not believe any report to the contrary, I repeat I am perfectly right, Clement." A few honrs later he felt very sick, and afterwards became unconscious and had an epileptiform seizure.

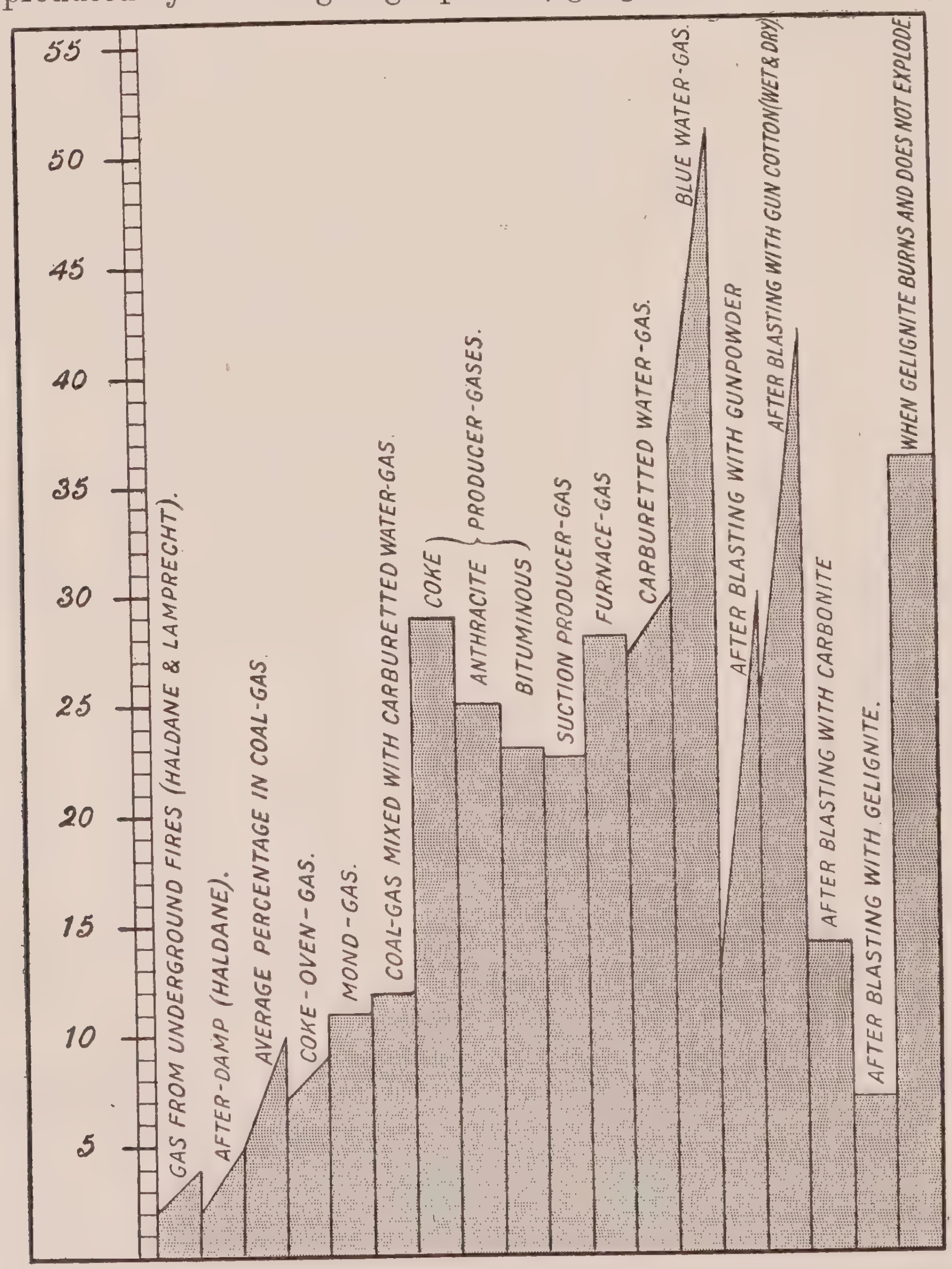
[1] Sir C. Foster & Haldane. "The Investigation of Mine Air," London, 1905, p. 176.

CHAPTER VIII.

POISONING BY BLAST-FURNACE GAS; VARIOUS PRODUCER GASES—MOND, DOWSON, SUCTION; ILLUMINANT, Etc.

FIG. I.

Figure showing graphically the percentage amounts of carbon monoxide present in after-damp, furnace gas, illuminant and producer gases, also in gases produced by the firing of gunpowder, gelignite, etc.



Note.—The shaded parts show the percentage of carbon monoxide.

From the annual reports of the Factory Department of the Home Office during the last ten years, it will be found that cases of poisoning by carbon monoxide are increasing. From 1899 till 1903 there were fifty-one cases, seventeen of which were fatal, and in 1904 and 1905, fifty-seven cases, eighteen of which were fatal.

TABLE XVI.

Nature of Gas.	Year 1906.	1907.	1908.	1909.
Carbon Monoxide . .	55	81	55	53
(*a*) Blast-furnace and furnace	17	37	26	16
(*b*) Power (Suction, Producer, Mond, Dowson) . .	20	16	19	25
(*c*) Coal, etc. . . .	16	21	10	12

Blast-furnace Gas.

In the old days when furnaces were open and where the valuable gases of combustion were allowed to escape into the atmosphere, the furnace-men who fed the furnaces, which was done from the top, were frequently exposed to the dangers of poisoning by gas. Often fatal cases occurred, and, moreover, there was a large number of minor cases of "gassing." But at the present day, where the top of the furnace is closed, cases of "gassing" at the furnace-top are very much less common, as the time taken up in lowering and raising the bell, the only mechanism whereat the gases are allowed to escape, is much too short to permit of serious poisoning, while in the most improved furnaces where, for example, the double-charging bell method is in use, very little gas can possibly escape. Of course where the bell or cap does not fit properly, gases do escape, and in this way serious poisoning, both acute and chronic, has occurred. Where the bell had been leaking, for example, the wind has carried the gas to a bell-man who was in his cabin and there asphyxiated him. Nor is it only gas poisoning which the furnaceman has to dread on the furnace top; there are also the deadly "slips" which cause so many fatalities. Automatic charging-plants are now largely in use in America, in Germany, in iron-furnaces near Middlesborough, and in other places, and as the men in these do not go to the top such accidents are prevented. In these plants the material is tipped automatically from the skip and the bell lowered by a contracting lever in the hoisting engine cabin.

Notwithstanding what has been said, however, a certain number of

cases do occur in various ways. One of the Factory Inspectors (Stockton District) declares that cases of poisoning by gas in iron-furnaces are very much more common than is generally recognised; for when the men are gassed they are very chary of reporting themselves to the management, as they are afraid, rightly or wrongly, that they might be deemed too susceptible to the gas and, consequently, that their services would be dispensed with. In support of his contention, this inspector quotes figures which have been supplied to him by an official in connection with a large Friendly Society, many of whose members are workers in the blast-furnaces in the Midlands, to the effect that *nearly fifty cases of slight poisoning by carbon monoxide were brought to his notice between January and August of 1906.*[1]

A modern blast-furnace gives off enormous quantities of gases, viz.—some 150,000 to 200,000 cubic feet of combustible gases per ton of fuel consumed. These passing through outlets near the top are carried to the "downcomer" and from thence through culverts to be purified. The gases are then freed from dust and scrubbed, so as to recover the most valuable bye-products such as tar, oils, etc., the ammonia contained in the gas being fixed by weak sulphuric acid to form a solution of sulphate of ammonia, which is evaporated down into solid sulphate of ammonia, a most valuable commercial product. What is aimed at in the blast-furnace is to produce iron by reducing iron ores with the help of carbon monoxide, which is produced by the combustion of coal or coke. The working ought to be regulated in such a manner that reduction is regularly and continuously going on. Naumann found as a result of his experiments that when coal is burned in air, formation of CO_2 begins about 400°C., and quickly increases up to 600°C. or 700°C. when the maximum is produced. Carbon monoxide in small amount (20 per cent.) is produced at this temperature, but this increases till 1000°C. is reached; when above this temperature all the carbon is changed into CO, no CO_2 being then produced.

TABLE XVII.

Showing Temperature relative to Production of Carbon Monoxide and Dioxide Gases.

	CO.	CO_2.
450°C	2·0%	98·0%
600°C	23·0%	77·0%
800°C	90·0%	10·0%
900°C	96·5%	3·5%
1000°C	99·3%	0·7%

[1] Factory Reports, 1906, p. 96.

With regard to the temperature and production of furnace-gas, valuable practical experiments have been performed by Schenck and others to determine the proportion of CO to CO_2. Below 450°C. very little CO is produced (about 2·0 per cent.), most of the gas being CO_2. Above 1000°C., CO only is produced.

As it is most important that we should know with what gases we are dealing in poisoning cases, analyses obtained from different sources are here set down (Table XVIII.) which may be looked upon as a fair guide to the composition of the gaseous products found in furnaces which are fed with different fuels, and in the following table (Table XIX.), a recent analysis is given of the gases from the furnaces in Coltness Ironworks, Lanarkshire, which was obtained from the analyst.

TABLE XVIII.

Gas.	Coke-fed Furnaces.		Coal-fed.	
	A	B.	C.	D.
CO_2	4·5	10·5	12·5	8·0
CO	36·0	23·0	26·5	28·0
N	56·0	59·3	58·5	55·0
H	2·5	6·8	2·5	5·0
CH_4	1·0	2·4	1·5	4·0
	100·0	100·0	100·0	100·0

TABLE XIX.

Analysis of Blast-furnace-gas in Coltness Ironworks.

Gas.	Unwashed Gas (Green-gas).	Washed Gas (Clear-gas).
CO_2	6·6 — 7·0	6·0 — 6·6
CO	27·4 — 28·0	27·2 — 28·2
N	55·5 — 56·5	55·6 — 55·86
H	6·77— 6·86	7·44— 7·66
CH_4	2·64— 2·73	2·68— 2·74
	98·91—101·09	98·92—101·06

In considering these analyses, it is not difficult to come to the conclusion that it is the carbon monoxide which is the important factor in

cases of poisoning by furnace-gas. The experiments carried out by Lorraine Smith for the Water-Gas Committee (1899) also proved beyond doubt that in coal-gas and water-gas the poisonous effects were due to the presence of CO, and that the hydrocarbons were not present in sufficient quantity in illuminating and producer gases to have any poisonous action. In 1901, the same observer carried out further experiments with Percy Hoskins, and found that benzene was present in such small proportion that it could not possibly cause symptoms of poisoning. As for ethylene, when the greatest care was taken to free it from CO, it was found, even when 72 per cent. of this gas was present, that the effects produced were very slight; and that in ordinary coal-gas there is no more than 3 to 4 per cent.[1]

The crude gas which comes from the furnace before treatment is called "green gas," and after washing "clear gas." The crude gas, when it escapes, can easily be detected by the eye owing to the particles, siliceous, etc., which it carries with it, and, moreover, it has a distinct odour. When the gas is cleansed from these particles and dried, as it is when used in gas-engines, it is quite invisible and practically odourless, and is consequently much more insidious when it escapes.

The residual gas is used for a large number of useful purposes. Its components are powerfully reducing, and therefore are capable of combining rapidly with oxygen, evolving much heat. This is taken advantage of in many ways. Most of the iron works in Germany and the United States and many in our own country have now-a-days electric power stations which obtain their power from furnace-gas, and entire rolling-mills are now being driven by electricity derived from the blast-furnace gas. In Coltness Iron Works for example, the gas provides steam power for the whole work, while advantage is being taken of it directly for gas-engines whereby increased power is obtained. The gas burns the iron ore briquettes in kilns, and is used as fuel in the enamel brick-work which is half a mile distant from the furnaces. It is used along with producer-gas to melt the steel in the large steel foundry; while it also distils the coal for the gas-works which supplies the village with gas. It is also employed for distilling the ammonia liquor and for other purposes in connection with the ammonia works, and for generating the supply of electricity to the whole works for lighting and for motor purposes, as electric cranes, etc. It also supplies with motor power the mills at the large cement plant where Portland cement is manufactured from the molten slag of the furnaces, while advantage of it is taken for various other purposes.

Regarding the enormous amount of energy in these furnace-gases, it

[1] Lorraine Smith & Percy Hoskins: "An Experiment on the Effects of Inhalation of Ethylene," *Journ. of Hygiene.* Vol. 1, 1901, p. 123.

has been reckoned that a modern blast-furnace could give about 20 H.P. per ton of pig-iron produced, after allowing a loss of 10 per cent. during charging operations, and 35 per cent. for heating the blast, etc. So that what was formerly called "waste-gas" and treated as a waste-product is now of the greatest possible value, quite sufficient indeed to supply power for converting the ore into steel products ready for the market. Although it is only but recently that furnace-gases began to be used directly for the generation of power in gas-engines instead of under boilers to raise steam, sufficient time has elapsed to show that this is by far the most scientific, efficient, and economic method of using the gas, for there is thus more direct and immediate conversion into work of the heat-energy contained in the fuel. In the steam engine, heat is transferred first from the coal to the water, and it is only when the water is converted into steam under pressure that it is able to perform work, while in the gas-engine the heat is carried directly into the cylinder of the engine in the form of gas, without passing through any intermediate stage.

It has been calculated that there are now considerably over 120,000 engines of all sizes in this country worked by furnace and producer-gas. So that, as has been well said, while the nineteenth century has been the era of the steam engine, the twentieth century will be that of the gas-engine; and the prophecy made by Sir Frederick Bramwell at the York meeting of the British Association for the advancement of Science in 1881 viz. :—that in fifty years the gas-engine would have superseded the steam-engine—looks like being fulfilled.

With regard to its use in gas-engines, and in order to obtain the greatest possible efficiency of combustion, the gas requires to be cleansed and dried. Gases which contain too much condensible vapour and which deposit tar on the valves and sides of the valve-chambers prevent efficient working. The gritty dirt in the gas from the blast-furnace was found to be a great drawback to its use in large gas-engines, and many appliances are at work to get rid of this grit. Formerly, and even yet in some of the Ironworks, the dust was ignored, and a single downcomer carried it to an underground gas-flue leading to the stoves and boilers. In these flues, boilers, and stoves, the dust settled and accumulated, with the result that the whole plant had at intervals to be put out of operation till thoroughly cleansed, and gassing cases were frequent during this operation. The advent of the gas-engine demanded a clean gas, as dust, etc., soon blocks up the valves of the explosion chambers; so that it is now recognised that all the gas from the furnaces should be properly cleaned. This is carried out by various methods. The important point for us, however, is that the gas thus cleaned is much more dangerous. Furnace-gas, for example, when used for the steel work after being washed, etc., is much more liable to cause insidious

cases of poisoning because of its invisibility and inodorousness, than the gas direct from the producer.

How cases of gas-poisoning may and do occur at or near blast-furnaces.

Since, as in the case of large Ironworks, the gaseous products from the furnaces are used for such a great number of different purposes and are led for such distances, opportunities for escape of the gas, with consequent opportunities for the occurrence of cases of poisoning in the immediate vicinity, are likely to take place.

At the Furnaces.

As we have already said, cases of gassing occurring at the top of the furnaces are not nearly so common now as formerly when the top of the furnace was open. Still a number of cases do occur every year. In July 1911, in Langloan Ironworks, a hoist engineman was overcome by gas at the furnace top, and died. Gassing may occur on the top of old furnaces which leak considerably.

We call attention to a point which does not seem to have been commented upon before. When a furnace is "charged" for the first time, gas very often escapes through the seams of the iron plates on the top of the furnace on which the men are at work. These seams are not absolutely air-tight, but become so in a very short time owing to the tarry products, soot, etc., sealing them. The gas which escapes during the first few days must contain a considerable percentage of CO_2, and also much moisture, as frequently the bell "lamp" or "torch," which is placed so that it may set fire to any gas escaping, fails to light it, with the result that considerable volumes of gas escape and may cause serious symptoms to appear in the men who are working there. The furnacemen realise this fact quite fully, and they always look forward with dread to their shift during those few days; and if they can manufacture a good excuse for then shirking their work, they take advantage of it. While at work, they feel very "bad," having racking headache, slight giddiness, with palpitation perhaps, and they come home at night absolutely worn out and complaining of severe headache. In some cases, where the gas is not readily dissipated, as where there is little wind, it may collect in such quantity as to overcome and render some unconscious.

A defective bell has often caused serious cases of gassing. In one district where gas poisoning had been going on for a considerable time, and where several men had been permanently affected (p. 231), it was found that the bell was defective. When the bell was made thoroughly

gas-tight, cases of gassing became very much fewer. Occasionally fatal cases of carbon monoxide poisoning occur in those who are feeding the furnace. One case will serve as an example.[1] One Sunday morning, a furnaceman in an Ironworks in the Midlands was alone on the top feeding a furnace. He was found unconscious. It was supposed, as he was a thoroughly experienced man, that the accident must have been brought about by carelessness, either by failing to fire the gases after charging the furnaces, or while the gases were escaping, that he had neglected to go to the windward-side.

In course of time the lining of a furnace becomes worn and gives way in places, and, owing to the enormous heat, fissures in the stonework develop, which allow the gas to escape. This gas afterwards takes fire and burns with the characteristic bluish flame; but if it escapes combustion, and if the fissure is far up the furnace, the men on the top run the risk of being gassed, more especially if there is no wind; while further down it may hang about confined places and give rise to gassing.

It has been said that poisoning may occur owing to the gas, which escapes during what is called the "blow-out" period at the end of slag-tapping operations, collecting in the vicinity of the furnace-bosh. Thwaite had analyses of this gas made, and he found over 1 per cent. of CO with 8 per cent. of CO_2 present, which percentage of CO is of course very dangerous. If there is any obstacle to the gases escaping properly, as, for example, where the exhaust is not working effectively and where cakes have formed at certain levels of the charge—the so-called "scaffolding"—there may be an accumulation of gas which is likely to be discharged into the open during the "blow-out" period. But it then appears as a whitish cloud which is intensely hot, and the men are only too eager to get out of its reach, because when inhaled it causes intense coughing and sneezing. We remember one man who inhaled a small quantity of it, and who sneezed at very short intervals for considerably over an hour so violently that it caused intolerable headache and considerable epistaxis. Thwaite's warning, therefore, against isolated workmen working near the bosh during slag-tapping, should not be disregarded.

A most unusual accident was reported from Middlesborough in 1900.[2] A furnace was being "blown in"; that is to say, a fire had been kindled at the bottom of the furnace and the blast turned on, and then the furnace charged till it was about full. While engaged in tipping a barrow, the barrow fell in and the workman after it. When his mate found that he did not come out, he jumped in after him, and both men were fatally poisoned by the fumes which found their way up through the ore and fuel by the blast.

[1] Factory Reports, 1906, p. 31.

[2] Factory Reports, 1901, p. 94.

Most of the cases of gassing about furnaces are found among men who are cleaning or repairing the culverts and gas tubes about the furnaces, or the tubes in the ammonia recovery work and its connections; or, again, they may be gassed while doing repairs about the exhausters, or cleaning or working about the flues. It was while repairing the fans inside an exhauster that a young engineer in Coltness was gassed. As this case is in many ways, perhaps, one of the most remarkable on record, we refer to it in detail later (p. 295). Many of the larger tubes, those, for example, over two feet in diameter, are lined with brick, and we have seen cases of gas-poisoning in the bricklayers who were engaged in re-lining the tubes. Cases of gas-poisoning frequently occur when the underground flues are being cleaned. Some ironmasters, recognising the dangers of the gas leaking and collecting about the flues, have had the flues erected above ground, supported by iron pillars standing in the open air. Where these are adopted, gas-poisoning is not known.

Neither is the danger of these underground flues confined only to the times when they are being cleaned; for if there is any leakage, the gas tends to accumulate in and also to percolate through the soil and to find its way into cabins, etc. Where, also, these flues are in any confined corners of buildings where the gas may collect, poisoning may occur. In an Ironworks in the Midlands an additional furnace was to be put in blast, and certain tubes had to be broken in order to allow of connections with the new furnace being made. A portion of a pipe was cut off and sealed from the rest and left open for a fortnight. Two men who were at work on this had occasion to go inside. They were found dead. It was discovered that owing to the hammering, some rivet-holes had leaked and allowed the gas to escape. Analysis of the air inside the tube showed between 1·4 and 2·0 per cent. of CO.

In another case, also in England, several men who were engaged doing some repairs in the interior of an old furnace were gassed. One of these died after an illness of several weeks. This furnace had been out of use for several days, and as large quantities of water had been used to "wash it out," it was supposed to be perfectly safe. The fact, however, of the water being used, while combustion was going on though in an incomplete manner, would involve the generation of large volumes of carbon monoxide which had not been dissipated. Preventive measures in such a case are self-apparent. A much longer period should have elapsed before these men were allowed to go inside.

During recent years, a large number of accidents which have taken place during cleaning operations about furnaces have been recorded in Germany. For example, one man was fatally gassed while cleaning out the dust-catcher in connection with the blast-furnaces. This had been ventilated for an hour and a half, all the covers having been taken

off and the pipe left open. The man was found lying with his face in the dust. In attempting to rescue him, two other men were overcome, but recovered. In another Ironworks in Bohemia, six men were engaged cleaning the gas-cleaning apparatus in connection with a furnace. Before they entered it, the gas was cut off by a water-seal which had formerly always worked very well. The whole plant had previously been put out of action. While two of the men were working inside, gas suddenly streamed in and rendered them unconscious. The four men outside endeavoured to help them, but they also were overcome. When rescued they recovered, but the others died. Examination of the apparatus showed that the valve in the water-sealing tank was not water-tight, and as there was little water in it, the gas was allowed to pass.

In Prussia, two men who were descending a culvert in connection with the gas-washing apparatus of a blast-furnace were gassed. This case is of interest in showing that where a large percentage of CO is dissolved in water, it may be given off with fatal results to those exposed to it. Another case, also in Germany, has an important practical bearing, as it shows that collections of dust in the gas tubes of the furnace may absorb gas in sufficient quantity to prove fatal for a considerable time after these tubes have been exposed to the air. A man was engaged cleaning out a pipe which had been out of use for three hours. By raising the thick dust he set free quantities of CO which killed him. It was proved that the valve had been securely closed, and that the covers had been taken off the pipes for ventilation.

Gassing may occur in many other ways. One most unfortunate fatal case occurred in Coltness Ironworks in the following way. One of the chemists in the laboratory, a lad of eighteen, was sent into the test-room, a very small room through which tubes from the ammonia work are led for facility of being tapped, in order to enable the chemists to find out whether or not a proper amount of the bye-products is being passed. This is ascertained by passing the gas through a meter and taking a sample. It may be necessary, also, to test for SO_3, the presence of which in the mixed gases has a corrosive action on the pipes. This lad had shut the door of this poorly-ventilated room, seated himself, and commenced to read while waiting for a certain period to elapse before reading the meter. For certain reasons he was not missed for four or five hours, and, when discovered, it was found that he had been dead for a considerable time, his body being still in the position as if reading, with a paper in his hands. The gas had probably escaped from some of the rubber connections, and, the room being very badly ventilated, it had collected and done its deadly work. A fan was thereafter installed in this room, and certain regulations framed which are calculated to prevent such an accident in the future.

Explosions of gas about furnaces.

Serious explosions may occur in and about furnaces owing to ignition of collections of gas. A rather unusual accident, which led to the death of one man and the serious injury by burning of five others, occurred in Coltness in 1908. These men were engaged during the night in taking down the lining from the inside of a furnace. They stood on a scaffolding and were working with naked lights. When the furnace was "damped down," the large tube connection with the other furnaces, which was about six feet in diameter, had been closed or "stoppered" by loads of sand placed over clay which was arranged in its place over iron plates. This stopper must have given way, for an outburst of gas occurred which the naked lights exploded. One man, precipitated to the bottom, died of fractured skull, the other five, who were all seriously burned, climbed to the top of the furnace where they were rescued. In some of the forms of gas-cleaning apparatus, in cases of "hanging" of the furnace, or sudden stoppage of the blowing-engine, the flow of the gas from the furnace may cease or become much reduced. When this happens, there is danger of air being drawn into the apparatus and gas tubes, and of serious explosions resulting.

"Slips" at Blast-furnaces.

Sometimes a collection of smelting material gathers in the interior of the blast-furnaces. These are called "scaffolds," and are dangerous, as they not only hinder the proper working of the furnace by interfering with the escape of gas and obstructing the blast, but they give rise to what are known as "slips." The crust on the surface, owing to the collection of the charges, becomes thicker, while underneath, owing to the burning and settling of the smelting and the withdrawal of the slag, a large hollow filled wlth gases is formed. A furnace in this state is said to be "hanging," and it causes a good deal of uneasiness to the furnaceman. When the roof, the caked surface, gives way and falls in, there is a great outburst of the combined gases, flames, ashes, etc., which on dull days may be seen and heard in the neighbourhood within a radius of a mile. We have seen a considerable number of fatal accidents caused by the furnacemen being caught on the top, the men being terribly burned.

We have been struck by the fact, and this is confirmed by the experience of others, that in some of the men who have been burned, the severity of the symptoms is out of all proportion to the extent of the injuries, and that patients have died although the extent of the burns did not warrant such a grave prognosis. We have suggested that some of these deaths may be the result of neuritis of the vagus nerve which has been caused by small burns in the upper air-passages involving the

nerve-endings. It may be added that these accidents appear to be greatly on the increase owing, perhaps, to the inferior quality of the ore and fuel used.

Percolation of furnace-gas, etc., through soil, etc., causing poisoning at a distance from the leakage.

Many cases are on record where the gas has travelled through crevices or cracks in the tubes, etc., and having passed through the ground has appeared with deadly results at varying distances from its source. A few of the most interesting and instructive cases will be illustrative.

In 1857, at Clayton Moor near Whitehaven, gas from the iron foundries found its way into a neighbouring row of cottages and attacked thirty persons, of whom six died.[1] At the time, the effects were attributed to H_2S, but it is now known that they were due to CO. In Percy's *Metallurgy*[2] there is the following case. The head engineer of the Dowlas works was found dead in his office, which had been built on a cinder-tip. About fifteen yards away there was a brick culvert which had been made through this cinder heap, and which conveyed the blast-furnace gases to the forge-boilers. It was only a few days after the gas had been flowing through this culvert that the accident happened, the gases having found their way through the bricked culvert and then through the cinder-tip into the office, which was found full of gas. In an Ironworks in the North of England, a boy of fifteen was found dead in a foreman's office in the neighbourhood of the blast-furnaces. As there was no connexion in the room with any gas pipe or flue, it was difficult at first to decide how the gas, for there was a decided odour of it in the room, had found its way in. Afterwards, it was discovered that the joint of an 8-inch cast-iron pipe conveying blast-furnace gas to an engine, which pipe was a few feet from the office and two or three feet below the level of the ground, was leaking, and that the gas had found its way through the earth into the office. In another case, two engineers, one dead and the other dying, were discovered in a cabin where they had gone to rest. This cabin was situated between two furnaces which were connected by an underground brick and stone flue, but the cabin stood about forty feet back from this flue. The only way in which the gas could have found entrance was from this flue through the soil and into the cabin through the floor. In a blast-furnace near Newcastle, two men on the night shift went into a cabin to eat

[1] Letheby: "The Poisonous Effects of Carbonic Oxide," *Lancet*, 1st March 1862, p. 220.

[2] Percy: *Metallurgy, etc.*, Vol. II., p. 531.

their supper. One was discovered dead and the other died in a few hours. A slight leakage of furnace-gas was discovered which had found its way through the brickwork.

The following account of an extraordinary series of poisoning cases is from the Factory Report of 1901 (p. 94):—The cases happened in dwelling-houses which had been built on made-up ground consisting largely of porous slag, these cottages being near large blast-furnaces which stood on the banks of the Tees. One night while a gale was blowing, three men, who had gone to bed in one of those houses in the best of health, were found in an unconscious condition. They all recovered. Two months later, a number of other cases, principally in women and children, occurred in the same houses. These were all recognised as being cases of gas-poisoning. The doctor in attendance came to the conclusion that the only way the gas could gain access to the buildings was through the brick walls of cellars underneath. He proved, by watching the deflection of a taper flame, that, during the ebb tide in the river close by, air was sucked outwards through the crevices in the cellar walls, and was blown inwards as the tide flowed. It was also found that a large brick culvert, for distributing furnace-gas, was near the houses some feet below the ground. Probably the pumping effect of the tide drove the gas through joints in the brickwork of the culvert and through the soil and thus into the houses. It only required a sufficiently high wind and tide, as was present on both occasions, to cause sufficient gas to accumulate in the cellars. A ventilating pipe was sunk into the soil, the cellars were properly concreted, and it was hoped that this would prevent any further cases of poisoning.

Sir Thomas Oliver mentions a case where, in a village, carbon monoxide derived from the combustion of shale travelled thirty feet through the soil and, entering two houses, led to the death of two persons. Under the title "underground fires" (p. 42) we have already described cases in which the gas from a fire in an abandoned pit travelled through fissures in the strata and appeared with deadly results in a row of cottages. Several workmen in a large boiler-shop complained of feeling ill, two of these being rendered unconscious. The next day two others were seized in a similar manner. The only thing that was observed was a slight smell which was perceived to come from a certain part of the floor. This was opened. A few feet below the surface was revealed a bed of smouldering fire several feet in depth and several yards long. The floor had been made up of cinders, breeze, and ashes from locomotive boilers, and right through this the blast-pipe for the smiths' fires passed.[1] This material had been kindled, and the blast had fanned

[1] Factory Reports, 1901, p. 911.

it into an incandescent condition which resulted in the production of carbon monoxide.

In 1906, at Pelton Fell, Durham, two elderly people, a man aged sixty-one and a woman aged seventy-three, living a few doors from each other, were found dead in bed one morning. The houses had been built near the edge of a ravine. A short time previously, the colliery owners had allowed refuse, consisting of shale and barytes, to be tipped into ground at the back of the houses. This refuse, probably as a result of spontaneous combustion, had caught fire about a month before, and the fumes had permeated the soil and found their way into the houses. Carbon monoxide was found in the blood of both. The following fatal accident is typical of many which occur annually. At Carriden, Bo'ness, in August, 1911, a man, thirty years of age, was suffocated by the fumes from a refuse bing where he had lain down to sleep.[1]

Many cases of poisoning by illuminant-gas are on record where people residing in houses, some of which had no gas connections at all, have been poisoned, the gas percolating through the sub-soil into the house from a broken gas-main, perhaps many feet away, in some cases as far as sixty feet. In some of these cases the gas found its way from a broken or leaking pipe into the houses through the drains. It has also been definitely proved by Pettenkofer and several German writers that gases can easily penetrate brick walls; indeed, given the thickness of the brick, the temperature, and the pressure of the gas, it is now found quite possible to estimate the volume of gas which will pass. It has also been found that the mortar which unites the bricks is even much more permeable than the bricks. Where the brickwork is defective, the gas will have less difficulty in finding its way through.

Percy,[2] in his standard work on Iron and Steel, records the following interesting case. A workman, returning home from the night-shift, found his mother, sister, brother, and a lodger, all dead. The house consisted of two lower rooms and two bedrooms. The north wall of the lower rooms was built against the side of the stalk of a blast-furnace. The same wall of the upper room was connected with the furnace by an arch and a quantity of brick-work, three feet thick. The house was twenty feet from the bottom of the furnaces. There was a crack in the furnace through which gas was passing. As there was not only a crack in the brickwork of the wall of the house, but the wall itself was very loose, it was not difficult for the gas to find its way into the house. In the Factory Reports, records will be found of several cases where the

[1] See also Glaister: "Carbon Monoxide Poisoning," *The Lancet*, 8th and 15th December 1901. *Text Book of Medical Jurisprudence and Toxicology*," p. 649, *et seq.*

[2] Percy: *Metallurgy*, Iron and Steel, p. 527.

gas had found its way from an underground flue to weighing-cabins or cabin-shelters, with serious results to the occupants.

In the old days, and even yet in some works, there were often dwelling houses not far from the furnaces, and as the foundations of these houses were sometimes not very good, the gas has been known to find its way into the houses; and near all furnaces there are cabins, weigh-houses, etc., in which men have been and are liable to be gassed. In offices and engine-houses also, where the foundation is not cemented, cases are on record where the gas has permeated the sub-soil and found an entrance.

In connection with this question of the percolation of gas through soil, we cite one case in our experience which might have ended more seriously than it did. In an engine-house, an engineer was sent down to pack the condenser-rod of the pumping engine. It was situated two or three feet below the floor of the engine-house. While removing the packing, he was suddenly overcome, but was dragged out by his mates, one of whom took his place. He also was overpowered, as were three others who, in turn, tried to finish the job. Before they could proceed any further they had to connect a bag-pipe with the compressed air (the blast for steel furnaces) and blow out the accumulation of gas. If one of these men had been working by himself he would probably have perished, as the gas was present in considerable quantity, if one may judge by the rapidity with which the men were overcome. In this case there was, in addition, owing to the confined space, a want of oxygen, the CO and other gases having displaced the air. The interesting point, however, is this, that the gas must have travelled a considerable distance, probably from some leaking gas-pipe. The gas had then probably found its way into an old conduit and old pipes which were underneath the engine-house, and then had easily percolated through the soil into the engine-house owing to the higher temperature. In the Prussian Factory Report of 1904, will be found an account of an accident where four men were killed by gas while pumping out an underground conduit in connection with a furnace. The gas had found its way into the conduit in some unaccountable way.

Another point to remember, by no means an unimportant one, is that furnace, illuminating, etc., gas during its passage through the soil loses to a considerable extent its characteristic odour, and men in engine-houses, etc., become so accustomed to the gassy odour round the works that their attention may not be specially attracted to its presence in increased amount. Moist earth will not readily allow the gas to pass, but round about furnaces, flues, and pipes, and the houses in the vicinity, the sub-soil is generally fairly dry and easily permeable by gas; and when the gas is heated its permeability is much increased.

'After being washed, furnace-gas is most dangerous from the fact that it has not a very aggressive odour, and, consequently, in and around iron-works where the air, as has been already said, is at all times tainted, especially near the furnaces and ammonia and steel works, its odour may not be recognised, with the result that insidious cases of gas poisoning arise. We have already mentioned, also, that for use in gas engines the gas requires to be cleansed and dried, that is, freed from suspended particles and moisture. These in the crude gas, as it escapes into the atmosphere, make it quite visible as a cloud, because vapour condenses on the particles of dust. But the purified gas, and, to a less extent, that which has percolated through soil, is practically invisible, and at the same time has lost its distinctive odour.

In connection with the percolation of gas through soil, it should be further noted that in many iron-works cases have happened where labourers, engaged in digging trenches, etc., in ground near waste-gas flues not absolutely air-tight, or near leaking pipes, etc., have been overcome by the gas. As the ground in the vicinity of blast-furnaces, etc., must be almost honeycombed with gas pipes from which leakages may occur, all buildings, cabins, weigh-houses, and engine-houses, should not only be very well ventilated by windows on the outside walls permitting through ventilation, but the ground should be covered by a layer of cement at least six inches deep, with an air space between it and the floor. Made-up ground for building on is decidedly objectionable. Where there is any danger of gas collecting in any building, there ought to be a scheme of artificial ventilation by fans; and in all cases where gas-engines are in use, no matter how small these may be, they should be housed in well-ventilated rooms.

The late Sir Thomas Stevenson[1] reported a very unusual accident of CO poisoning at Leeds Forge Works in 1889, which resulted in the death of two forgemen. These men were found dead in a cabin which was lighted by two burners and warmed by a cooking stove, water-gas being used for combustion. The products of combustion were allowed to escape into the room. The tap supplying the gas stove was found partly turned on, but not lighted. No smell of gas was detected. The post-mortem examination was conducted by Dr Scatterwood, his son, and Dr Hargreaves. Three other surgeons and two students were also present. While the examination was proceeding, Dr Scatterwood's son collapsed, while Dr Scatterwood himself felt ill. Then one of the students and one of the doctors collapsed and fell down, while all the others were slightly affected. It was then discovered that eight gas jets were partly turned on, and yet no odour of gas was detected. The accident caused a good deal of excitement amongst the workers, as it was thought that

[1] Thomas Stevenson: Guy's Hospital Reports, 1889, p. 221.

these cases of poisoning had resulted from breathing the emanations from the dead bodies.

An extraordinary and unusual accident occurred in 1909 at the Hoerde Works in Westphalia. As a safety ventilator belonging to a pipe-conduit containing gas was not working properly, the order was given to allow the gas to escape. When let off, the gas streamed along a street quite near the furnaces, found its way into dwelling-houses, and poisoned over forty persons. Fortunately the accident was immediately discovered, and the victims were removed to the hospital in connexion with the works.

Gassing in or near boilers fired by furnace-gas.

A large number of ironworks use a considerable proportion of their furnace-gas under boilers for steam-raising purposes; and a substantial number of cases of gas-poisoning, some of them with a fatal termination, have already occurred among men engaged in cleaning the flues and scaling the interior of these boilers. There may be a range of six to twelve boilers one of which is periodically stopped for cleaning. The gas to the boilers passes through a regulating valve, which is fitted to the boiler front, and is then directed, mixed with a proper proportion of air, into the flues. When a boiler is put out of action, it is necessary to see that the gas-valve is absolutely shut, for if there should be any leakage through the valve, it can readily be understood that a man working inside a narrow flue in a confined space would soon be fatally gassed. One of us [1] had experience of such a case in a public work in Glasgow. A man was engaged temporarily to "scale" and clean the interior of a retort, one of a series, which was supplied by producer-gas. The retort had been out of use for some time. The man began operations about 6.30 a.m. When the signal for breakfast was given at 9 a.m., he did not emerge from the retort, whereupon the foreman, thinking that he had not heard the ordinary signal, knocked loudly on the outside of the retort with a hammer and then left for his own breakfast. On the foreman's return from breakfast, he was informed that the man in the retort had not yet appeared, whereupon search was made inside. The man was found therein lying dead, his naked lamp burning not far from where his body lay. We made a post-mortem examination for the Crown the following day, the chief facts revealed at the necropsy being as follow:—The body was that of a vigorous, well-nourished man about middle life. Over nearly the entire front aspect of the external surface of the body, the skin bore the characteristic rosy-pink coloration of carbon monoxide gas poisoning, while internally there was the usual pink suffusion of the

[1] Glaister: "Water-Gas, Carburetted Water-Gas, and Carbon Monoxide Poisoning," *The Lancet*, 8th and 15th December, 1906

surface of the intestines, and the arterial hue of the blood. Spectroscopic examination of the blood proved the presence of carboxy-hæmoglobin, and quantitative estimation that the blood was saturated with CO gas to the extent of 75 per cent. A Fatal Accidents Inquiry was held, at which we gave evidence, and the jury thereafter returned a verdict that death was caused by "suffocation from gas fumes." Men should never be engaged singly at work of this kind, and one person should be detailed to supervise and be responsible for the safety of the other inside.

The following are further examples of how gassing may be produced. In 1904, in an iron-works in the Midlands of England, two men were engaged in cleaning a boiler, one of a range of eight supplied by furnace-gas, and which had for the time been disconnected. After getting inside, they told their apprentice to shut the furnace door. Shortly afterwards, a heavy "kick" occurred in the blast-furnace, which slightly displaced the dampers cutting off the furnace from the boiler, with the result that the gas made its way into the boiler and fatally poisoned the men. At the Clyde ironworks, a workman entering the repair shop shortly before 6 a.m. found two men lying unconscious. They died shortly afterwards. The shop in which the bodies were discovered was situated quite near the boilers which were fired by gas. There had always been an escape from these, and it was found that this was not any greater than usual. What happened was that the wind, which was blowing in the direction of the shop at the time, carried the gas there and gradually asphyxiated the men.

Men Gassed while working in Perilous Positions.

Recognising the characteristic action of carbon monoxide as causing powerlessness of the limbs long before, perhaps, total loss of consciousness supervenes, it can readily be understood that men are sometimes placed in most perilous positions by being gassed. They may be working, for example, on platforms placed at considerable heights from the ground near a leaking tube, or on roofs of engine-houses, under cover of which gas has collected from a burst pipe. While a man was doing some repairs on an overhead crane in a Steelwork, he felt himself getting giddy and "queer." He understood from his symptoms that he was being gassed, but was able to step from the crane to the top of the blast engine-house beside which he happened to be at the time, where he collapsed. Had he not been an experienced man and recognised that he was being overcome by the gas, and had he remained on the crane, even although there was a rail round it to guard against falling, the probabilities are that he would in his helpless condition have fallen over the rail to the ground and been killed. The men who went to his rescue also felt the effects of gas-poisoning.

In July 1909, while two steeplejacks were working on a narrow platform at the top of a chimney stalk, 180 feet high, in Coltness Ironworks, one of them became unconscious through inhaling the poisonous gases issuing from the stalk. The other, feeling himself being gradually overcome, resolved to come down for help, but before doing so he strapped his unconscious companion with a rope to the two narrow planks, about nine inches broad, which composed the small platform surrounding the stalk and on which they were working. When he descended he collapsed, and became unconscious. Two men, David McWhirter and Wm. McClelland volunteered to go to the aid of the man at the top. To reach him they had to climb a vertical ladder 180 feet high, and the man lay at the opposite side. They succeeded in carrying the unconscious man round to the ladder, placing him in the "boatswain's chair" used by steeplejacks, and in lowering him in safety to the ground. For this daring rescue these men received the King Edward Medal of the First Class. Two fatal cases, where men working in dangerous positions were gassed and fell to the ground, occurred in 1911 at Birmingham, in each case death being the result of the injuries received by the fall. In feeding the hopper, while standing on the top of a producer plant, a man was overcome by the fumes, and falling to the ground a distance of eight feet, received a fracture of the base of the skull from which he died

Illuminating-Gas.

As it will be necessary from time to time to compare cases of poisoning by illuminating-gas with those by furnace and producer-gas, we have here inserted analyses of illuminating-gas (see Table XVIII). In illuminating-gases the amount of CO present differs considerably in different samples. In some cases where it consists practically of blue water-gas, sometimes as much as 50 per cent. has been found, while others contain as little as ordinary unmixed coal-gas.

Ordinary coal-gas is made by heating coal in air-tight retorts (long fire-clay tubes), where it is subjected to great heat by a furnace underneath, the gas being abstracted from the retorts by an exhauster. The crude gas is purified by various methods. It is first cooled and condensed, when the tar separates. The gas now passes to scrubbers, which consist of large cylinders filled with coke through which a stream of water passes constantly, or washers, whereby any tar that is left is removed, and with it the ammonia. It is further purified by removal of carbon dioxide gas in ordinary lime purifiers, of sulphuretted hydrogen, and of carbon bisulphide, etc., over the oxide bed. In coking plants and gas works which do not contain processes for the recovery of the sulphur,

the waste gases contain a relatively large percentage of H_2S. This, as we have already seen, is a poisonous gas, and the greatest care is therefore required in disposing of the waste gas. After the gas is washed and the ammonia removed, it passes through sulphuric acid and, lastly, over the oxide bed, which is in the open. The gases removed by purification methods are composed of 16 parts H_2S, 81 parts CO_2, and 3 parts HCN, per cent. The oxide bed is generally a small structure from 4 to 6 feet deep, and built of brick, the floor being fitted with grids on which the oxide rests. A fatal case of gassing, where death was due to H_2S poisoning, occurred in a gas-works where the victim was employed in cleaning out and renewing the oxide in one compartment while the other was in use. Accidents also occur in the lime purifiers, when sufficient time has not elapsed with the covers off to allow of the escape of noxious gases.

Pure Water-Gas (*non-carburetted water-gas, blue water-gas*) is made by passing steam over incandescent coke. As this gas has no smell, and burns with a bluish non-luminous flame, it is necessary to enrich it with oil (hydro-carbons) which is vaporised and mixed with the gas, with the result that the flame is improved, being now much more luminous, and possessing a decided odour. The gas so treated is now called *carburetted water-gas.* As it still contains a very high percentage of carbon monoxide, it is generally mixed with coal-gas in such proportions as to make the mixture less poisonous. In this country about ten per cent of carburetted water-gas is found in ordinary coal-gas.

The highest proportion of CO in the enriched gas used by many gas works is 17 per cent. and a steady average may be taken as 12 per cent. Formerly some Companies used as much as 50 per cent. water-gas, but it should never be more than 25 per cent. and preferably should be even less.

The Departmental Committee, appointed in 1898 to enquire into the manufacture and use of water-gas and other gases containing a large proportion of carbonic oxide, issued their report in 1899. They recommended that the manufacture of any poisonous gas for heating and lighting should be prohibited unless the gas possessed a distinct and pungent smell, that the quantity of CO in any gas should be limited, and that Companies manufacturing such should keep a record of the amount.

Acts carrying out these recommendations have since come into force. Gases having a large percentage of CO now require to be made odorous, and the amount of CO present to be limited to 14 per cent. If the gas contain more than this, the Secretary of State is required to impose regulations to protect against the risk of poisoning, the Inspector of Factories to enforce the provisions where the gas is used in factories and workshops.

The amounts of CO in the principal illuminating gases are, respectively, as follows :—

Non-carburetted water-gas	40 to 50% CO
Carburetted water-gas	27 „ 30% CO
Coal-gas (depending on kind of coal and conditions of manufacture) . . .	4 „ 12% CO
Coal-gas mixed with carburetted water-gas (average)	9 „ 12% CO

Regarding the dangers from gas containing a high percentage of carbonic oxide, we have simply to add that these arise only when the gas escapes unburned, since, of course, the higher the percentage of CO in the gas the greater the danger. When properly consumed, non-carburetted water-gas is no more poisonous than coal-gas.[1]

Coke-oven Gas.

Coke is necessary for certain metallurgical processes; for example, large quantities are used for iron smelting, etc. Generally with coke-ovens the coke is the main product, the gas the bye-product. Many collieries have now erected these in which to utilise the slack and small coal, and in an increasing number of them they are utilising the valuable bye-products, such as tar and ammonia. There is also a large number of collieries which use the spare gas from their coke-ovens under steam boilers for giving power to winding-engines, air-compressors, pumps, etc. The example of the iron works in using the furnace-gas in gas-engines has been followed by a number of collieries in order to utilise these gases more perfectly; and it is found that sufficient power is generated to supply the greater part of the colliery, besides being a very great saving in consumption of coal. Coke-oven gas is very similar in composition to coal-gas, as the method of manufacture is the same, but it is not purified.

TABLE XVIII.

Analyses of Coke-Oven Gas and Illuminating-Gas.

	Coke-Oven Gas.	Illuminating-Gas.	Dellwik-Fleischer Process.	
			Water-Gas.	Blue Water-Gas
H	47 —55%	49·0%	49·0%	51·0%
CH_4	27 —32%	32·5%	0·7%	1·0%
CH_2	3·0— 3.5%	1·5%	5·0%	4·5%
N	8 —12%	1·4%	6·3%	3·5%
CO	6 — 8%	8·8%	39·0%	40·0%

[1] Glaister: *Lancet, op. cit.*

The quantity of nitrogen in coke-oven gas may be high if there is any leakage of air through faulty joints. In illuminating-gas the amount of CO_2 is generally small, as means are adopted to remove it. Coke-oven gas is used in many iron and steel works for firing steam boilers and also for use in gas engines. At these coke-ovens, more especially in connection with collieries, several fatal gas-poisoning cases have been seen, where tramps, in their quest for a warm sleeping-place, lie down beside the ovens and the leaking gas has asphyxiated them.

Gas-Poisoning and Explosions in Gas-Works.

We have already seen that accidents sometimes occur in gas-works while the purifiers are being cleaned, if sufficient time has not been allowed to elapse for the noxious gases, H_2S, CO_2, and CO, to escape. Leakages of gas often occur in gas works, giving rise to carbon monoxide poisoning. At the Wrexham Gas Works, owing to an over-pressure of gas, the water-seal at the valve connecting the purifiers was forced, with the result that there was a sudden out-pouring of the gas. The under-manager went down into the cellar under the purifier, and in attempting to stop the pipe was overcome.

Owing to the large number of gas-poisoning cases and explosions which occur about gas-works, particular attention is now being paid to the type of purifier houses, the methods of emptying them, the locating of escapes of gas, etc. Purifier houses which are not properly ventilated should be condemned. Where the side walls have not sufficient openings, accumulations of gas are apt to occur which may give rise to explosions. In these houses a very important question is the proper lighting. Where the best type of house exists this is not so important, as the gas readily escapes and the light is good. Electric torches are generally used, and all naked lights should be prohibited; but even electric light has occasionally given rise to explosions. In fact, it is better to do without lights altogether inside those houses. They should be lighted from the outside. The building should be lofty and roomy, with all sides open. By far the best type is that built on the open shed principle, *i.e.*, with iron pillars only supporting the roof In this way accumulations of gas would be prevented, and the vexed question of how to light these houses would also be settled.

Gas Poisoning from "Geysers."

Many fatal cases of carbon monoxide poisoning have occurred, a few in our own country, but many more in Sweden, Denmark, France, and Germany, from the use of badly constructed "geysers," or "instantaneous heaters," for baths in ill-ventilated bath-rooms. In many of these

cases the deaths have resulted when the victims were in the baths, as in the following case which was recently reported by Ryberg.[1] A young woman was found dead in a bath with her mouth under the water. The room, which was very small, was completely closed, one gas bracket being still lit, but the geyser was extinguished. There was no smell of gas. Experiments proved that when the stove in this small, closed room was lighted, so much oxygen was consumed that the gas burned badly and was sometimes extinguished. From this it was supposed that, owing to the deficiency of oxygen, the woman had first been rendered unconscious and then drowned, but examination of the blood showed the presence of a considerable amount of CO. After an experiment in which the stove was kept burning for nineteen minutes in the closed bath-room, the analysis of the air showed 2·5 per cent. CO_2, 13·9 per cent. oxygen, 1 per cent. CO; and the poorer the atmosphere became in oxygen the larger would be the resulting percentage proportion of CO. In this case there was no pipe to carry away the bad air. Ryberg holds the opinion that the law should insist on proper ventilation wherever "geysers" are to be used. Such laws are now in force in Hamburg and in Sweden. We also have had experience of similar cases.

Poisoning by Various Producer-Gases.

The use of producer-gas has enormously increased during the last ten years, and everything points to its being still more largely extended. The following is a list of the chief industries in which it has been employed.

Mond Gas and its Applications.

MOND GAS IS BEING USED :—

In Iron and Steel Works :—

For melting steel in open hearth furnaces.
„ heating billets in mill furnaces.
„ heating steel ingots.
„ forging and re-heating furnaces.
„ annealing and tempering armour plates.
„ puddling iron.
„ plate-bending, flanging, and dishing furnaces.
„ annealing iron and steel sheets, wire, etc.
„ heating ladles.

[1] Ryberg: "Om ett fall af Koloxidförgiftning och om faran af Gasbadugnar *Meddelanden f. Läkaresälls*, 1902, p. 274, ref. in *Nord. Med. Ark.*, 1902, p. 40.

In Foundries:—

For core stoves.
,, drying moulds in pits, on bogies, or in suspension.
,, firing crucible furnaces for melting brass, steel, malleable iron, etc.
,, heating ladles.
,, annealing castings.

For Smelting:—

For roasting and calcining ores.
,, refining copper, zinc, etc.

In Shipbuilding and Engineering Works:—

For heating plate and angle furnaces.
,, heating, forging and annealing furnaces.
,, core and mould drying stoves.
,, case-hardening furnaces.
,, heating galvanising baths.
,, heating smithy furnaces.
,, firing boilers.

In Glass Works:—

For melting glass in pot or tank furnaces.
,, annealing ovens.
,, power for rolling, polishing, etc.

In China, Earthenware, and Fireclay Works:—

For firing kilns for earthenware, porcelain, and china-glazed ware.
,, firing kilns for glazed fireclay articles, bricks, tiles and sanitary ware, baths, etc.
,, firing kilns for ordinary fireclay goods, such as bricks, slabs, ornamental tiles, etc.

In Metal Works:—

For heating brass and copper in muffles.
,, heating tubes in muffles.
,, annealing, tempering, and hardening.
,, case-hardening and japanning.
,, heating enamelling ovens and stoves.
,, brazing and other operations where high local heats are necessary.

In Galvanising Works:—

For furnace work of all kinds.
,, roasting ores.
,, distilling and evaporating liquors, tar, oil, varnish, etc.
,, electro-chemical operations.

In White Lead Works:—

For litharge furnaces.
„ lead-melting pots.

In Salt Works:—

For evaporating brine.

In Textile Mills:—

For singeing and gassing yarn.

In Dyeing and Dressing Works:—

For heating large drying-room.
„ power.

For Power Purposes:—

In electro-chemical works.
„ textile machinery works.
„ railway workshops and docks.
„ central electric stations, for power and lighting.
„ electric tramways and railways.
„ flour mills.
„ paper works.
„ collieries and mines.

What is aimed at in these producers, of which there is a large variety on the market, is to convert, as far as possible, solid fuel (coal, coke, or coal-slack) into combustible gases. The fixed carbon which the solid fuel contains is turned into CO_2, and the aim is to convert the whole of this gas, if possible, into CO. By passing air over red-hot coke, CO is produced; thus $C + O = CO$, and any CO_2 which is formed becomes decomposed by the hot coke into CO.

Thus: $CO_2 + C = 2CO$.

This is what is called simple producer-gas. Its drawback is that it has a very low calorific power. It was therefore necessary to obtain a better gas with a less loss of heat. Now if a steam blast is passed over red-hot coke the following action takes place, viz.:—

$$C + H_2O = CO + 2H.$$

The difference between this and simple producer-gas is that although the same amount of CO is produced, in this way there is, in addition, an equal volume of combustible hydrogen, while in the other there is only the inert gas nitrogen. But the decomposition of the water or steam absorbs heat which would very quickly cool the fuel below the temperature at which decomposition would take place, and the action would cease. To do away with this, therefore, a mixture of air and steam in proper proportion is blown in, with the result that the combustion of the carbon by the air produces sufficient heat to equalise the absorption of the heat by the decomposition of the steam.

From the producer the gas passes by suitable ports or openings to the downtake, whence it is conveyed by culverts or pipes. In Siemen's original furnace the gas was supplied to the furnace cold, and so the producer was at a distance from the furnace with a long cooling tube overhead. But most works supply it hot, and therefore have the producer close to the furnace, using underground flues. It is when cleaning these underground flues in steel and other works that so many cases of gassing take place. In producer-gas made from anthracite in a Dowson plant, there is more hydrogen (19·25 per cent.) and slightly less CO (20·1 per cent.) than in the gas produced from coke (H, 14·3 per cent.; CO, 22·5 per cent.).[1]

TABLE XIX.

Average Analyses of Various Producer-Gases and Furnace-Gas.

	A. Producer-Gases.			B. Furnace-Gas.	
	(*a*) Siemen's	(*b*) Dowson	(*c*) Suction	(*a*)	(*b*)
CO	24·4	18·20—25·07	25·0	25·0	30·0
H	8·6	18·73—26·55	18·8	7·9	6·8
CO_2	5·2	6·57—11·30	6·8	12·1	8·5
CH_4	2·4	0·8 — 1·4	0·5	1·2	2·2
N	59·4	42·28—48·98	49·0	53·8	52·3

Mond gas is perhaps the most largely used of the producer-gases, being employed on an enormous scale in some districts as Lancashire, where it is conveyed in large pipes to a great number of works.

Combustion is carried on at a comparatively low temperature in order to provide against the destruction of the ammonia, and also to

TABLE XX.

Analyses of Mond Gas. (Power Gas Corporation).

	A. Mond Gas from Bituminous Coal with Ammonia Recovery.	B. Mond Gas from Bituminous Coal without Ammonia Recovery.
CO	11·0%	23·0%
CO_2	16·5%	5·0%
H	27·5%	17·0%
CH_4	3·0%	3·0%
N	42·0%	52·0%

[1] Dowson and Larter: "Producer-Gas," p. 24.

prevent formation of clinker in the producer. In order to effect this a blast of hot air and steam is used. In connection with Mond plants, gas-poisoning often occurs during the repairing and cleaning of the towers of the apparatus.

Mond gas differs, then, from most producer-gases in composition, as owing to the greater use of steam in the process, it has a much higher percentage of hydrogen and CO_2 and considerably less CO.

The gases CO, CH_4 and H_2 are combustible, and the virtue of the combined gases depends upon the quantity of these present; Mond gas depending upon its large amount of hydrogen, while in most producers a large percentage of CO is aimed at, as some maintain that this gas has a relatively higher efficiency as a furnace fuel.

How accidents occur in steel works where producer-gas is used, and also in connection with various producer plants.

A large number of cases of gas-poisoning have been seen in connection with Steelworks where the open hearth or Siemen's process is used. In this method gas is used as fuel. This may be obtained straight from iron furnaces, some works depending upon this supply augmented, perhaps, by producer-gas, or obtaining their entire supply from producers. The blast-furnace gas is regarded by the men as being much more dangerous and insidious than producer-gas. As steel works are more shut-in than iron blast-furnaces, which stand in the open, probably most cases of gas-poisoning occur there. There is always a small percentage of gas about steel furnaces where gas is used as fuel, and the men working there sometimes complain of feeling giddy and unsteady whenever they come out of the shop into the open.

Many accidents occur when the underground flues, gas-box, valves, and tubes are being cleaned. When they are cleaning the "buttons" out in the open, if a wind is blowing the operation is usually safe, but if not, the workmen may be quickly gassed. It cannot be urged too strongly that gassing can quite readily take place in the open. This can easily be understood if the peculiar cumulative action of carbon monoxide be remembered, as every inspiration of the CO-laden air throws out of action numerous red blood corpuscles. But it is principally in underground flues, and when the "gas-box" is being cleaned, that poisoning takes place; and here it would be a good plan before allowing the men inside, to drive out the gas by a blast of compressed air.

We have already drawn attention to a case where the wind drove gas, which was used for firing boilers, into an engine-house, and which resulted in the death of two men. A similar case occurred in connection with a steel work which led to the gassing of a large number of men, but

happily without serious results. The accident was brought about by a burst gas-tube outside the shop, and the wind driving the gas into the shop, the unusual spectacle was seen of men staggering to the door and then falling unconscious outside, and of others dropping to the floor inside, at short intervals. Within the same shop a leaking tube led to the insidious poisoning of several persons in its immediate vicinity. These men felt themselves getting ill, left their work, and went outside, but whenever they got out, their symptoms became very much worse, partly owing to the exertion of walking and partly owing to the cold atmosphere. Some of them staggered about holding their heads with their hands from severe headache and were giddy, while some sank to the ground in an unconscious condition.

In connection with producer plants, the management often encounter the greatest possible difficulty in getting their workmen to realise how dangerous is the gas with which they are working; and many accidents are brought about by gross carelessness, just as accidents in connection with gas-engines are produced. In 1904, in a pipe foundry in Glasgow, where a Dowson gas plant was used, the man who looked after it was found dead one morning beside the scrubber. It was found on examination that an overflow pipe from the scrubber had become corroded at the water-seal, and that the *leak had been stopped by a piece of cotton waste.*

A considerable number of gas-poisoning cases have occurred through men cleaning out the Dowson tanks before the gas had been allowed to escape thoroughly. In all producer plants accidents occur where these are not housed in properly ventilated sheds, the gas readily collecting where ventilation is defective. As there is always the danger of small leakages from joints and fittings, these plants should always be in the open air, with only a roof-covering to protect the engineman from the weather. Where the plant is in the open, opportunities for gas to collect do not occur, and there is no chance of gas finding its way into factory rooms and living-rooms, as has so frequently happened and given rise to gas poisoning. In the Dowson gas producer plant, poisoning has occurred among those who have to go into the syphon pit for stoking purposes, the syphon pit being often a few feet, four to six perhaps, below the ground level, and approached by steps. Gas from leakages sometimes accumulates here, and has caused gas-poisoning in the attendants.

One rather unusual case of poisoning, ending fatally, occurred at Oldham, where a man was gassed while engaged in unscrewing a plug at the lowest part of a tube near the ground level to let out the water which collected there. So much gas escaped when he unscrewed the plug that he was overpowered before he could put it back, and when found, he was unconscious and inhaling large quantities of the gas, which was escaping freely. On no account, therefore, should any part of a pro-

ducer plant be entered before the gas has been completely expelled. Gassing cases sometimes occur also through clearance pipes for producer plants being inside the building. These should always be taken outside the building, and be fixed to end a few feet above it. Where they end within the building, serious explosions have occurred owing to the gas being ignited by naked lights. In cleaning out the fire of the generator of a producer plant in Edinburgh, a man who was working alone was poisoned, and lost the use of his legs. As he did not come home, enquiries were made, and he was found. *In any work like this, which entails the risk of gas poisoning, men should be prohibited from working alone.*

Gas poisoning in iron foundries.

Sometimes the men who are engaged in charging the cupola from the raised platforms are overcome by gas, such cases occurring much more readily when the cupola is inside the shop. A workman was fatally gassed by venturing, although warned not to do so, into a cupola furnace to do some chipping after it had been lit. He was in but a very short time, and was got out with all speed, but he never regained consciousness. In another case, after the cupola has been charged and lighted, a man jumped in at the charging hole to stop a small crack with clay, although he had been warned not to do so. He was immediately overcome, and his mate, who went to his rescue, was also rendered unconscious, as was also a third man. The three men were got out with the greatest difficulty. The first two died, and the third was dangerously ill for some days. In another similar case two men, standing on a stage thirteen feet from the ground, had been charging an open cupola blast-furnace in the ordinary way, through an opening about twenty inches square. In the absence of his fellow-workman, the other, thinking too much metal had been put in, thrust his head into the furnace and removed a quantity, but after pulling out two cwt., was overcome, and was found dead in this position by his mate when he returned.

Gas Engines.

A considerable proportion of cases of carbon monoxide poisoning occurs in connection with gas-engines. With those engines, however, which obtain their supply of gas from the ordinary town supply, cases of poisoning are uncommon. But such may occur where there is any leakage and where the room in which the engine is placed is badly ventilated. For gas-engines run by illuminating-gas, india-rubber gas-bags with metal casings which act as reservoirs are supplied. The rubber sometimes cracks, with the result that gas may escape into the room. A fatal accident occurred in 1908, where the occupier of a small factory

was found dead in his gas-engine room. The gas-pipe from the town supply was found disconnected, with the gas turned on. It is chiefly in connection with suction-gas engine plants, however, that the larger percentage of cases of poisoning by carbon monoxide takes place.

We shall, first, shortly describe these plants and how they are worked; next, discuss rules and regulations which are often issued by manufacturers of such engines by reference to cases of poisoning which have occurred; then draw attention to the manner in which these do occur; and, lastly, describe various precautionary measures which should be adopted when such gas-engines are installed.

It was at first thought that with suction-gas plants very few cases of poisoning would occur, seeing that there are no chambers which require to be entered for cleaning purposes, and if there chanced to be any leakage, that air would enter the producer rather than that the gas should escape from it into the room. But this has been found to be very far from the case. On the contrary, more cases of poisoning probably arise from suction producer plants than from any other gas-producing apparatus. The suction-gas engine has become very popular, and is now extensively used for many purposes, owing, probably, to the fact that it takes up little room, is cheaply and easily run, and does not entail, comparatively speaking, a large initial outlay. The plant, too, may be of any power, low or high, is very simple in construction, and, as it is quite automatic in action, is easily looked after if the directions issued by the manufacturers are faithfully carried out. Most plants require neither steam-jet nor air-blower (although some types still have the latter), the air being drawn into the producer by the suction or charging-stroke of the gas-engine which tends to create a vacuum in the producer, hence the name "suction engine."

The plant consists of (1) a generator made of steel plates lined with fire brick, on the top of which is a stoking valve and hopper of the rotatory type, so arranged that it is impossible to admit air during stoking; (2) a vaporiser; and (3) a gas purifier or scrubber, all of which are coupled up direct to a gas engine. The gas in this type is taken directly to the combustion chamber of the engine, and as gas is only produced while the engine is actually working, no gas holder is required. The vaporiser, which takes the place of the boiler in other plants, may be on the top of the producer in the form of a water-jacket; in the larger forms it is external but quite close to the producer, in the form of a tubular vaporiser or evaporator. Steam is generated by this vaporiser by utilising the waste heat of the producer and of the gas. With each charging-stroke of the gas-engine, gas is drawn into the cylinder at a few lbs. below atmospheric pressure, with the result that air is sucked

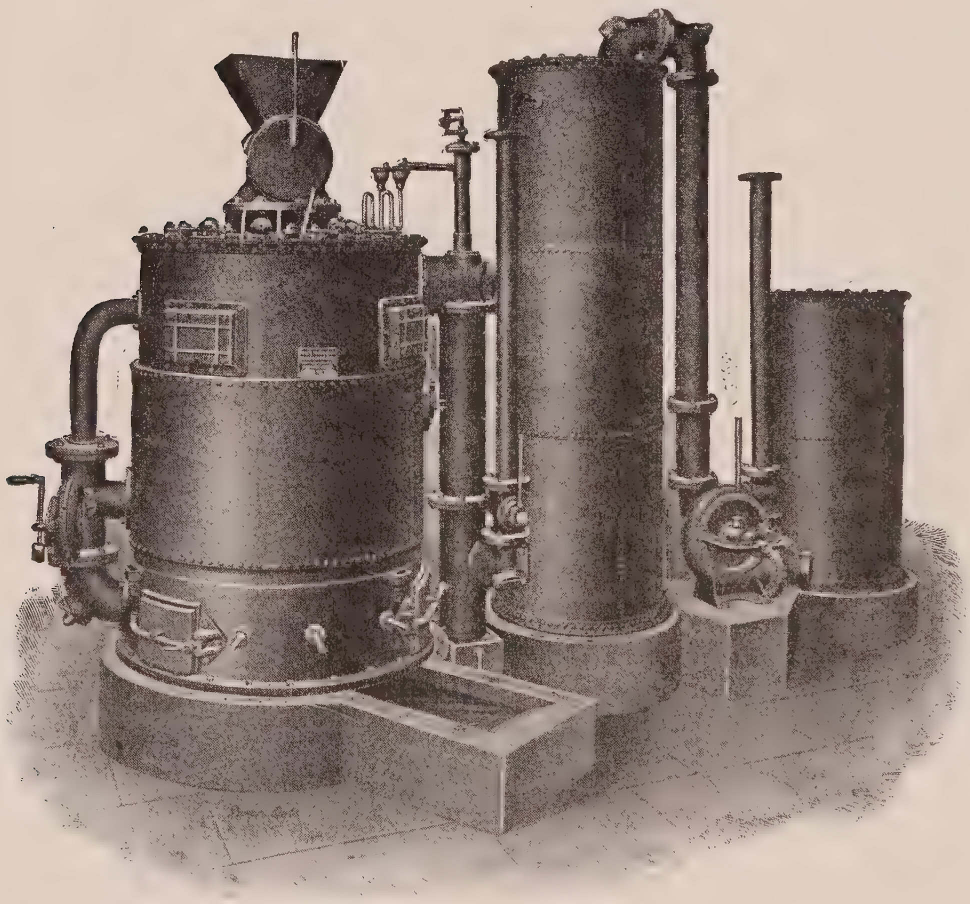
in through air-inlets situated at the top of the vaporiser. This air charged with steam as it passes over the hot water, passes down the steam and air-pipe, and becomes hotter and hotter as it is exposed to the high temperature. Passing over the incandescent fuel, the steam is decomposed into hydrogen, carbon monoxide, etc., as has been already described.

Fig. II.

PATENT "SUCTION-PRESSURE" GAS PRODUCER.

For Heating, Furnace Work, Engine Driving, etc.

(Anthracite, Coke, or Charcoal.)



GENERAL DESCRIPTION.

The plant, as illustrated above, consists substantially of a generator (*a*) in which the fuel is burned and the gas generated; washer (*b*) containing coke filling through which the gas is then passed, cooled and cleaned; and a dryer, or scrubber (*c*) by means of which moisture and also the last traces of dust and other particles in suspense are removed from the gas before its passage to the engine.

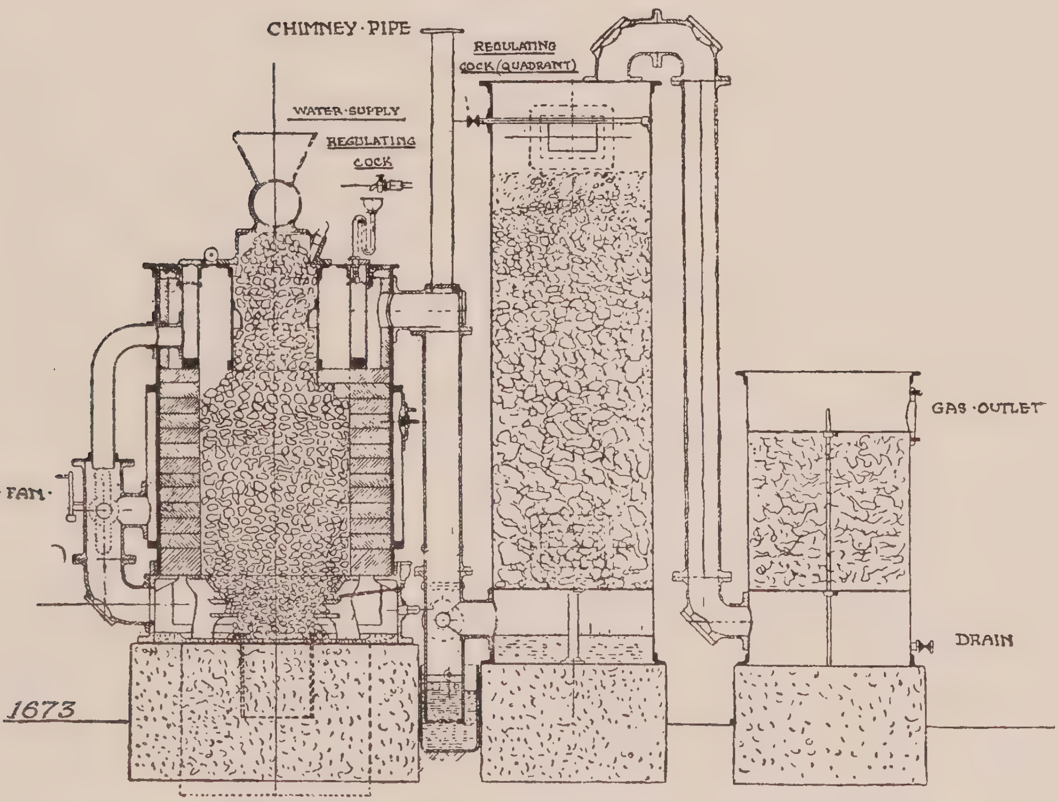
The following is an analysis of average gas so produced.

TABLE XXI.

Suction-Producer Gas.

	A	B
CO . .	20·7 per cent.	22·9 per cent.
H_2 . .	24·6 ,,	20·4 ,,
CO_2 . .	7·5 ,,	4·6 ,,
N . .	46.4 ,,	51·7 ,,
O . .	0·8 ,,	0·4 ,,
	100·0	100·0

Fig. III.



Patentees and Makers—THE POWER-GAS CORPORATION LTD., STOCKTON-ON-TEES.

This gas now passes through the scrubber to the engine, where it meets with more air, and is exploded in the cylinder.

As regards the exhaust gases, these under normal conditions are harmless, as they consist of the products of complete combustion of producer-gas. But when combustion has been incomplete, CO, acetylene, with, perhaps, some of the original gas which has escaped combustion, are present as well as the more innocuous products of complete combustion.

From what has been said, it will be apparent that when the engine is at work, leakage into the open is not likely to happen, as then the whole plant is under suction or partial vacuum; hence, should there be any defect in the plant, such as defective joints, air would be drawn into the plant.

The following are precautionary notices issued by the Gas Corporation Coy. for the guidance of the workman using the plant.

Leaks.—"To keep the plant in good working order and also to avoid danger, any leak must be immediately stopped. If this cannot be done while the plant is running, then it must be shut down."

Leakages may occur through the ignition tube, for with tube-ignition small quantities of unburned gas escape at each stroke. Leakage may also occur through the water-seal being forced or where it is empty, but there are now appliances on the market to prevent this. Gas may escape during the compression-stroke through a leaky engine-piston. Whatever the source, leakages are constantly occurring in gas engine plants: it may be from the cylinders, or from faulty connections and valves. In one work two men were gassed by leakage from a tester-cock. Leakage of the burned products past the exhaust valve stem is often serious, as analysis of the gas shows large proportions of CO. To detect leakage is very important, and several contrivances are now on the market for this purpose. One of the simplest is to fix a small cup containing water over the valve, which is placed uppermost, so that bubbles of gas escaping can be seen quite easily, and thus an empty cylinder at the critical moment prevented. Leakages give rise to very serious results, more especially where the engine-room is badly ventilated. In a Salford factory, in the early morning, seven women who were working in a room immediately above the engine-room were overcome by the gas, which had accumulated in the engine-room below, and found its way up. There may also be leaking in the generator. And when the glands of a double-acting gas-engine or the pistons or air-valves are not tight enough, leakage will take place into the engine-house itself. As the gas contains a large percentage of CO, even a small escape may occasion serious poisoning.

Starting.—"At starting there is no suction, but there is a pressure of gas owing to the starting-fan being worked. So that, at that time, if there is any hole or leak, instead of air leaking in, the gas will leak out into the engine room or producer house"

"Take care, when starting, that you have main stop-valve closed and chimney-valve open."

In reviewing a considerable number of cases of poisoning in connection with suction-producer plants, we have found that it is chiefly during starting operations that the principal danger arises. When the engine

is not working, the coke in the generator is kept at a dull-red heat in order to economise fuel. In order to start the engine, air is forced through the fuel by means of a "blower" in some plants, or by a fan in those of a better and more modern type, till the gas is burning well at the test-cock. Then the blower or fan is stopped, and the engine started in the usual way. The engine then, by its own suction action, draws the necessary amount of steam and air through the fuel, thus producing its own power-gas. Now, during this starting operation, which may take from 10 to 25 minutes, there is no suction, but the plant is under pressure, so that should there be any leakage or holes, or defective fittings or joints, the gas will find its way into the engine-room. During this process the chimney valve should be open, so that the products of combustion may escape by a waste pipe. Should the attendant start the engine with the crank a complete revolution ahead of the correct position for starting, the gas and air valves will both be open, and with the fan or "blower" at work, the gas, blown through the engine, may pass through these valves and through the air-box into the engine-room. Starting the engine, then, with the valves in the wrong position, may lead to serious poisoning. For example, in one case reported by the factory inspectors in Germany, the engineer in charge of such a plant, in replacing a three-way-cock which had been taken out to be cleaned and repaired, turned it the wrong way by 180°, with the result that the motor could not be started. While looking for the cause of this, the blast-cock was turned on so long that the producer-gas, which was now present in large amount, rushed through all the connections of the gas pipe, as the gas was now under pressure, and poisoned all the workmen who were round the motor.

Testing.—Test-cocks are generally fitted to the plants for testing the quality of the gas before starting the engine.

"All test-cocks must be looked at to see if they are closed. When using a test-cock to test the quality of the gas, if the gas does not light up the cock must not be left open, but turned off until required to test the gas again. But no light must be applied after erection or after cleaning out the plant, until all air has been blown out of the washers and pipes."

It not infrequently happens that the attendant, when he finds that the gas is not strong enough to burn, does not take the trouble to screw off the cock, but allows the gas to escape into the room. In no case should this be done; the cock must always be screwed off. On no account must the waste gas be used for testing purposes, as serious explosions have occurred from this cause.

Stopping.—"As soon as the engine gas stop-valve is shut, immedi-

ately open the valve in the chimney pipe, so as to allow a free way for the gas to the outside air. This is very important."

Cleaning.— Gas poisoning cases have frequently arisen among workmen cleaning the plant, more especially the scrubbers.

"Whenever the Plant is going to be cleaned, it must be entirely stopped, and the fire must be completely out. Air should then be blown through the Plant by a fan. The generator ash doors should then be opened, and after this the Plant must be left alone for several hours."

"The doors and windows of the Producer house should also be kept open."

"All cleaning should be done by daylight; and there should be no fire, nor light, nor smoking near the Plant. Also there should always be two men when cleaning-out has to be done."

"The piston of any gas engine, and especially when producer-gas is used, should be kept in good order, and no waste gases be allowed to escape, as some of the producer-gas may escape when the engine is running. If the engine blows at the cylinder front when running, the piston should be removed and the fault put right for safety as well as to avoid loss of power."

Two or three fatal cases of poisoning have occurred recently where the men entered the scrubbers too soon after opening. In these cases, the fact of the engine-houses being very badly ventilated also contributed to their death, as the gas could not properly get away.

Air-Intake.—The intake-air for the engine should never be taken from the inside of the engine house but directly from the air outside.

"It is advisable to have the engine air-intake placed in the open air, so as to avoid any chance of gas getting into the engine-room through the air-box. In certain circumstances and positions of the valves the gas could do so, and the attendant is therefore warned that it is possible for gas to escape in this way."

Charging.—"When charging the hopper with coal, do not place your head immediately above the hopper, and avoid breathing any gas that may be in it. Also when poking the fire, keep your face away from the door to avoid any flame that may shoot out."

Ignoring this warning has led to serious accidents. For example, the case we now quote resulted from negligence, and occurred in spite of previous instructions given and a copy of the Factory Memorandum left in the engine-house. The workman was just going to start the engine and had given the fan a few turns, when there resulted a light back pressure in the producer. He then climbed to the top of the producer, which was about 7 ft. high, and opened both the lower and upper slides of the hopper in order to put in the coal, and while doing

so the back pressure caused the gas to escape. He was overcome, fell off the producer, and fractured the base of his skull from the fall.

It is also important, as serious accidents have occurred from this cause, to see that the waste-pipe is carried out into the open air and clear of all windows of work-rooms, etc. An interesting case, showing how gassing may occur, resulted in the loss of eight valuable horses, which sickened one after the other, and died. Here the gas plant was placed outside in a yard, but the exhaust from the engine, instead of being carried beyond the roof, opened into a covered passage in which the horses had to stand while the drays were being loaded. The engine, being new, was not working properly, hence probably there was an increased quantity of CO in the exhaust. In another case, the exhaust pipe of the engine discharged directly into a cotton mill, and led to the gassing of four operatives. In another factory, the waste-pipe for blowing off the gas when starting the suction-gas producer was found to be discharging into the air of a work-room, fourteen feet above the floor. In all these cases the pipes should have been made to open into the open air a few feet above the roof of the factory. In another case, in a boot factory, half-a-dozen women and two men employed on the first floor were gassed. It was found that a gas producer had been erected in the open air on the side of the factory nearest to the work-room, and the waste gas not consumed by the engine was allowed to blow off, some of which had probably been carried by the wind and found its way through the ventilator. After the accident, arrangements were made to burn the waste gases. In Germany, a case was reported by the Factory Inspectors in which a workman, who was in a w.c. in a mill, was poisoned by suction-gas. It was found that the gas-pipe between the cleaner and the motor, instead of being carried out on the roof, had been allowed to open outside at the level of the floor of the w.c. which had been built over it.

Under no circumstances should exhaust gases be allowed to discharge into conduits, etc., in works, or into sewers. Neglect of this led to the following accident. Two men who were working in a culvert in a work died, after several hours' illness, from the effects of what was regarded as CO poisoning. The only possible source of the mischief was the gases discharged from the exhaust-pipe of a gas engine which was at a distance from the place where the men were at work. The exhaust pipe opened directly into the culvert. This case came under the notice of Délèpine, who investigated the matter, and carried out most interesting experiments to demonstrate the danger from the gases discharged by the exhaust pipe of gas engines when working under certain conditions. Carbon monoxide gas can only be found in the exhaust gas when combustion is imperfect, and when there is too little gas in the mixture of gas and air supplied

to the engine, the mixture does not ignite, with the result that unburnt producer-gas, with its large percentage of CO, is found in the exhaust. Up to a certain point the richer the supply of gas, the greater the speed of the engine, but if there is too great a proportion of gas to air, the speed is reduced.

The following is Délèpine's summary of the results of his experiments :—[1]

"1. When the engine works normally, *i.e.* with a proper supply of gas and air and with a load that does not affect the speed materially, the exhaust gas, though containing some CO, is clearly noxious to rabbits and guinea-pigs only after exposures of more than one hour.

"2. When the engine is supplied with a proper amount of gas and air, but is loaded to such an extent that it is nearly stopped, the slightly diluted gas produces in guinea-pigs severe symptoms of asphyxia in four or five minutes.

"3. When the engine is supplied with a full amount of gas, but when the air is reduced so as to lower the speed considerably, the exhaust gas becomes poisonous to guinea-pigs in less than half a minute.

"4. When the speed of the engine is reduced owing to an insufficient supply of gas, the exhaust gas does not materially affect guinea-pigs so long as the air-supply is proportional to, or in excess of the gas supply."

From the foregoing it would appear, therefore, that the exhaust gases are particularly dangerous :—(*a*) when the gas supply to the engine is excessive ; (*b*) when the gas engine is heavily loaded ; and that the former condition is more dangerous than the latter. Délèpine found acetylene present in considerable amount in certain samples, and in such, CO gas was also present in large quantity. As acetylene has a distinct odour and also irritating properties when inhaled, it would act as a danger signal of the presence of the deadly CO. He demonstrated that CO and not acetylene was the poisonous agent in poisoning by exhaust gases.

From his experiments Délèpine concluded :—

"1. That the main cause of danger when my experimental engine was over-loaded was the considerable reduction in the amount of oxygen contained in the exhaust gas ; the proportion of carbon dioxide was also very large. The danger began to be rapidly manifest when the quantity of exhaust gas exceeded 50 per cent.

"2. That the main cause of danger when the supply of air to my experimental engine was insufficient, was the presence of a large amount of carbon monoxide. This danger began to be manifest when the proportion of exhaust gas reached 1 part of gas to 40 or 50 parts of air.

[1] *Public Health*, May 1911.

The CO present in the exhaust gas is partly derived from the unburnt coal-gas, and partly from the imperfect combustion of the gas that is utilised. It is probable, therefore, that the use of power-gas is attended with greater danger than the use of ordinary lighting-gas. Anything slightly blocking the entrance of the air-pipe is sufficient to bring about this danger."

Délèpine noted when the gas supply to the engine was full and the air-supply reduced to a minimum, that "the smell of gas as it escaped from the exhaust pipe suggested the presence of impure acetylene and of products of combustion of oil. This gas had an irritating action on the mucous membranes of the nose and of the eyes. After staying a few minutes in the neighbourhood of the exhaust pipe," he says, "I felt giddy and suffered from some unsteadiness in my movements, and one of my assistants felt the same symptoms. After washing the gas through water and passing it over lime, I found that it had lost its irritating properties, but I was then able to detect clearly the smell of coal-gas. The purified exhaust gas was poisonous to animals in the same way as the unpurified gas; the products of the oxidation of nitrogen and sulphur were not, therefore, the source of danger." This gas on analysis was found to contain 4·47 per cent. of carbon monoxide. Experiments were made upon mice with this gas undiluted and untreated, and it was discovered that the animals showed collapse or unconsciousness at the end of 20 seconds, and died in less than half a minute.

From these experiments, therefore, it is obvious that the chief cause of danger arises when the supply of air to a gas engine is insufficient, thus giving rise to the abundant formation of CO in the exhaust gas, partly derived from unburnt coal-gas and partly from imperfect combustion of the gas used; hence any obstruction in the air-pipe of an engine would produce this danger.

In Germany, as in our own country, a large proportion of the cases of industrial carbon monoxide poisoning have been reported in connection with suction gas-engines. A serious case was caused by a man in charge of a gas-engine plant opening a valve too soon while two men were working in the pit underneath, with the result that they were overcome. The engineer managed to rescue them, but another man who had gone to their rescue was rendered unconscious, and the same fate befell the head engineer and two of his assistants. All attempts to get these men out proved unsuccessful; indeed, during the efforts to rescue them four others were gassed, but afterwards recovered. When the first four were rescued, life was found to be extinct in all.

Plants which are driven by gas under pressure have also contributed largely to fatalities in Germany. An accident occurred in a linen factory

in Berlin where such a plant was used for heating the irons. The waste gas was extracted by suction. No CO was at any previous time found in the ironing-room, but one day, owing to the mechanical ventilation being put off by mistake, twelve women were seriously affected.

In nearly all gas-engine rooms, it is found that the men in charge for the first two or three weeks complain very much of headache, giddiness on exertion, being "off their usual," and being utterly worn-out when they arrive home at the end of their day's work. This feeling, caused by the inhaling of small quantities of gas, and which is always more or less present, soon wears off as toleration becomes established. The same occurs where the engine is in a factory or shop in which a number of persons are working, viz.—the newcomers usually suffer for some time. The greatest care, then, should be taken to carry away all gas, and where there is any difficulty about this, a scheme of mechanical ventilation should be installed, and, if necessary, a ventilating shaft should be run up from the fan to the roof of the factory or shop, with the producer out in the open. Where the plant is put into a basement there should always be mechanical ventilation.

The following preventive measures are advised in the Memorandum issued from the Home Office as to the use of Water-Gas, Suction-Gas, and other Gases in Factories (April 1910.):—

1. Notices should be posted up stating the deadly nature of the gas, the symptoms produced by its inhalation, and the best means of rendering aid to those who are gassed.

2. Persons in charge of any engine worked by water-gas, or of any apparatus in which it is stored, or otherwise exposed to risk of inhaling CO, should be free from any disease of the heart or lungs. Employers would do well to cause persons about to be so employed to be examined and certified as fit by a medical man.

3. No engine in which water-gas is used should be in a confined space.

4. A competent and responsible person should at stated short intervals inspect all valves and connections to see that there is no escape of gas; and a signed record, with the dates of such inspections, should be kept in a book for that purpose.

5. The openings giving access to any part of the water-gas circuit should be few and in positions as safe as possible, and should be opened only in cases of real need and by responsible persons.

6. No workman should enter or approach, when opened, the holder or other part of the water-gas circuit until the gas has been well flushed out by fresh air.

The following has been issued by The Power Gas Corporation, with

the approval of the Home Office, to be affixed in a prominent place in any building in which a producer-gas plant is installed :—

NOTICE.

Breathing of Producer-Gas should be avoided. It is dangerous when breathed in quantity.

SYMPTOMS. The *first* symptoms produced by breathing the gas are, *giddiness, weakness* in the legs, and *palpitation* of the heart.

If a man should feel these symptoms, he should *at once* move into *fresh warm air*, when in slight cases they will quickly disappear.

Exposure to cold should be *avoided* as it aggravates the symptoms.

A man should *not* walk home too soon after recovery, as muscular exertion, when affected by the gas, is to be *avoided.*

MEDICAL AID. If a man should be found insensible or seriously ill from the gas, he should *at once* be removed *into fresh warm air*, and immediate information be sent to the Oxygen Administrator, *a medical man being sent for at the same time.*

REPAIRS. *No man should work alone on any work which would be likely to involve exposure to the gas. Should the nature of the work cause the man to enter a culvert or hole, he should have a rope tied securely round his waist, held at the other end by his mate standing outside.*

NIGHT WORK. On any occasion when the Plant is required to work through the night, the Watchman should visit the Plant on each round, and his call should be answered.

THE OXYGEN CYLINDER. The cylinder should be provided with a lever key, nipple and union, together with a rubber tube at the end of which is a mouthpiece. It is also advisable to have a small pressure gauge attached to the cylinder so that loss of Oxygen may be observed and the cylinder kept in working order.

METHOD OF ADMINISTERING OXYGEN. Open the valve gradually by tapping the lever key (fully extended) with the wrist, until the Oxygen flows in a gentle stream from the mouthpiece, then place the mouthpiece in the patient's mouth and allow the Oxygen to be breathed until relief is obtained. The lips should not be closed round the mouthpiece, as it is important to

allow free egress for surplus Oxygen. The nostrils should be closed during inspiration or inflation of the lungs, and opened during expiration or deflation of the lungs, so that the Oxygen may be inhaled as pure as possible through the mouth.

If the teeth are set—close the lips and one nostril. Let the conical end of mouthpiece slightly enter the other nostril during inspiration and remove it for expiration.

Artificial Respiration is sometimes necessary in addition to the Oxygen inhalation if the Oxygen does not appear to act quickly.

Place the patient on his back, slightly raising the shoulders with a folded coat; remove everything tight about the chest and neck; draw the tongue forwards and maintain it in that position. Grasp the arms just above the elbows, and draw them steadily above the head, keeping them on the stretch for two seconds and then folding and pressing them against the chest for the same length of time. Repeat these movements about fifteen times a minute for at least half-an-hour, or until natural breathing has been initiated, when the Oxygen inhalation alone will suffice.

After recovery Oxygen inhalation at intervals should be continued as desired.

Poisoning by Charcoal Fumes.

Cases of accidental poisoning by charcoal vapour are recorded very frequently, and cases of suicide, more especially in France, are still deplorably frequent. On the Continent the extensive use of stoves for heating purposes, more especially the slow-combustion stoves of the Choubersky type, has been responsible for a considerable proportion of the accidents, the toxic gases escaping into the rooms owing to defects of construction, etc. In 1901, a defective stove was the cause of a terrible catastrophe in the hospital of a private asylum near Paris. Seven of the nine patients in the ward were found dead in the morning, and of the survivors one died on the following morning. Cases of poisoning have occurred from time to time by the fumes finding their way through faults in a chimney into a neighbouring house, with fatal results to the occupants. From the insidious nature of the poisoning in such cases, one can readily understand that in the case of a person found dead in a house in the morning, grave errors in diagnosis might be made and the death put down to natural causes.

The deaths of several important personages in Paris, among others

the famous novelist Zola, from the use of the stoves, drew the attention of the public authorities of Paris to the matter, and in 1902 the Prefect of Police nominated a Committee composed of engineers, hygienists, and architects. The duties of this committee were somewhat novel.[1] If any tenant were to complain about any noxious smell from the chimney or any other part of his house, and the landlord held that there was nothing wrong and that no alterations were necessary, it was to be the duty of the Committee to inquire into the matter, and if they found the complaints justified, to ask the landlord to carry out the necessary repairs. If he failed to do this, the Committee had powers to order the work to be done and to charge the expenses to the landlord. Since the dangers of the slow combustion stove were pointed out, the number of accidents has considerably diminished, as the type of stove on the market has been considerably improved, and accidents are now found chiefly arising from gross carelessness.

In the East, especially in India, a large number of poisoning cases occur from charcoal vapour during cold weather, owing to the natives using coke fires to heat their ill-ventilated huts. The coke is burned in any open receptacle, such as a kerosene oil tin. Natives regard the deaths as visitations from the spirits of the air.

Attention has been drawn to the dangers arising from banking-up fires for the night. Where a room is badly ventilated, and where owing to deficient chimney-draught the movement of air through the fire is much slowed, the result may be the formation of CO in considerable quantity, and it may find its way into the room atmosphere, and cause poisoning. "A large fire made to burn slowly and dully by the admixture with ashes, etc., by which the passage of air is retarded, is always risky, and if the chimney fails, is dangerous."[2]

Every now and again a case of gas poisoning occurs on board small ships, and these are doubtless attributable to the ignorance and carelessness of seamen. The sailors' cabin in the forecastle of a ship is often heated by a small cooking-stove or charcoal brazier, and the small outlet for the poisonous air may be closed by the occupants of the room when the weather is bad, and as the room is generally very small it immediately becomes a veritable death-trap. The following is a recent example. In 1911, at Greenock, ten members of the new torpedo-boat destroyer "Nautilus" were found lying in an unconscious condition in their bunks. It was found that they had been poisoned by the fumes of a coal stove left burning overnight in the forecastle.[3]

[1] *The Lancet*, 19th Jan. 1901, p. 219.

[2] *Brit. Med. Journ.*, 3rd March 1894, p. 483.

[3] See also cases in Glaister's paper on "Water-Gas, Carburetted Water-Gas, and Carbon Monoxide Poisoning," *The Lancet*, 8th and 15th December 1906.

With regard to the production of CO from the burning of charcoal, it is found that the quantity is very variable, the amount depending upon the activity of combustion, the rule being that the lower the combustion the larger the percentage of CO produced. Leblanc[1] analysed the air of a room which killed a dog in two minutes, and found 0·54 per cent. of CO. Eulenberg,[2] who carried out a large number of experiments with CO, found that the average amount of CO and CO_2 in eight analyses of charcoal vapour was 2·54 per cent. and 24·68 per cent. respectively. Biefel and Poleck[3] in their analyses of eight samples found much less, viz:— 6·75 per cent. of CO_2 and 0·34 per cent. of CO.

In considering the different action of charcoal fumes and illuminant-gas, it must be remembered that it is not only the amount of CO present which must be taken into consideration, but also the amount of CO_2, not omitting to note the diminution of the oxygen percentage. Many years ago Claude Bernard called attention to the fact that the presence of CO_2 in considerable amount intensified the action of CO, and from the physiological action of CO on the blood, it is not difficult to understand that a diminution in the oxygen percentage will also increase the action of CO. In charcoal vapour poisoning, therefore, a smaller percentage of CO may have fatal results, for there is not only less oxygen in the atmosphere, but there is a change in the proportion of the CO to the oxygen; and from the experimental work of Gréhant, Lesser, Haldane, and Mosso, we already know that the greater the percentage of oxygen in the air, the greater the quantity of CO is required and the longer time needed to produce fatal results.

Accident at Kilns (iron-ore briquette, brick, coke-ovens, cement works, lime works, etc.)

Many cases of poisoning have occurred in workmen at these kilns, especially those in which gas is used as fuel, and also among tramps who have been attracted to the vicinity by the alluring heat. Several cases have occurred at kilns in brickworks owing to leakage, the gas finding its way through fissures and cracks which are so often found in kilns and coke-ovens, and against or near which these vagrants lie for warmth. We must not forget, also, that tramps are generally in a condition which makes them fall an easy prey to carbon monoxide; they are often badly nourished, shivering with cold, poorly clad, and, perhaps, soaked with alcohol, all of which weaken their resisting powers to gas poisoning.

[1] Leblanc: *Annales Chemie et Phys.*, t. v., 1842, p. 240.
[2] Eulenberg: *Die Lehre von den schädlichen und giftigen Gasen*, 1865.
[3] Biefel and Poleck: *Zeits. f. Biolog.*, Bd. XVI., 1880, p. 279.

We remember one such case, viz., of a tramp, twenty-eight years of age, who, gaining access to a work, found his way into a small room in connection with the iron briquette kilns. In the floor of this room there was an escape of gas from the large pipes which supplied the kilns with gas. When found, the man was quite unconscious and breathing stertorously. The workmen insisted that he could not possibly have been in the room more than half-an-hour, and from the appearance of his face we should say that poisoning must have taken place rapidly. The workmen had removed him immediately into the open, which in this case was at the highest and most exposed point in the works. There they performed artificial respiration with the man's chest exposed—without any provision to restore and to sustain the body warmth. He was found about 4 a.m. and the morning was probably the coldest experienced that year, the wind being particularly cold and piercing. The reason these details are given is that they form an object lesson of how not to act in such a case. We have no hesitation in saying that the end was probably hastened, if not, perhaps, brought about, by the exposure to the cold. The man's body did not, however, exhibit the rosy or pink colour sometimes seen, as probably asphyxiation was too rapid. The face, in fact, had a dirty greyish colour, froth was exuding from the mouth when the men found him, but this had been wiped away; the eyes were staring, with the pupils widely dilated, and there was paralysis of the sphincters, urine and fæces having been involuntarily passed. He must have inhaled a very large quantity of gas, as the smell of gas in the expired air from his lungs when artificial respiration was being performed was so strong as to make one suppose that it might actually have been set on fire.

Two other cases (one of a tramp) having been gassed in the same Ironwork in much the same way, have come under our notice. A workman passing a bench found a tramp lying under it. He routed him out, telling him that there was a bad leak of gas there which came from a leaking gas-pipe. The workman himself then deliberately lay down in the place vacated by the tramp, and when found was dead. The other, a tramp, was found lying against the kilns in the briquette work, where he had gone for warmth. He also was dead when found. At Annan, the body of a man was found dead in the morning on the top of the lime kilns, where he had apparently gone to sleep. When the fires were put on in the morning he was asphyxiated.

The following case, which took place in Glasgow, was reported by one of us.[1] A tenement property, consisting of two flats occupied by a number of families, had been built within some inches of a lime-burning works, which consisted of three kilns. One night the family living in

[1] Glaister: *Medical Jurisprudence and Toxicology*, 1910, p. 652.

the ground floor apartments in that part of the tenement which was against the work, complained of a disagreeable smell, and soon after they became so ill that they thought it advisable to leave the house and take refuge in a neighbour's house. The people who resided in the flat above, however, were not so fortunate, for when the neighbours, alarmed by their non-appearance in the morning, entered the house, a woman and two children were found lying dead in the back apartment, and a man, who occupied the front room, was found in an unconscious condition. He recovered.

It is not uncommon for men who are doing repairs about kilns to be gassed even while they are out in the open. A fatal accident occurred in a cement work near Glasgow in the following way. A man while passing the top of a burning kiln was gassed and rendered unconscious. As he fell, he dropped a paraffin lamp he was carrying, which set fire to the gas, with the result that he was severely burned as well as gassed. The Factory Inspector found that the ventilation on the top of the kiln was very defective. At a cement work near Rochester, owing to neglect in testing the kilns and the flues of a number of kilns which had resumed burning after a lapse of thirteen months, a foreman and two workmen were fatally gassed, while four others who took part in their rescue were seriously affected.

Regarding such possible accidents the *Associated Portland Cement Manufacturers have issued* warning notices in which there is the following :—

"Regular inspection of kilns must be made on opening after being burnt off to see that they are safe for men to work in. Under normal conditions the kiln is partly, and sometimes entirely, drawn before the chamber is cool enough to enter to clear for reloading, and inspection must cover safety not only as to heat but as to gases. The eye in front of kiln and back eye of chamber must be opened when drawing is commenced, and entrance to chamber must be made cautiously. Should there be the slightest indication of gas, a paper torch must be thrown into the kiln and seen to burn out properly before work therein is commenced. If after several attempts it is clearly shown that a paper torch will not burn freely, the men must not be allowed to enter, and the matter must be at once reported to the manager. This applies more particularly where there is a kiln burning next to one that is being drawn, but in any case the dampers of the kiln being drawn must be down tight, and precautions taken generally to see that fumes from a burning kiln on the same flue cannot get back into a kiln in which men are at work, and this applies not only to the work of clearing or drawing, but to repair or any kind of work done in or about kilns."

In the case of a kiln which has lain cold for a long period, all the

above-named precautions must be observed, and, in addition, before men enter the pan or chamber, the drawing-eye of the kiln must be opened, and thoroughly freed below so that the air may pass into the charge. Employees are especially warned against adopting the means employed by many persons ignorant of the first principles of resuscitation, viz., placing men on their faces with their mouth over a hole in the ground. All such means are strictly forbidden. The administration of stimulants in any form is most dangerous, and is also strictly forbidden. [1]

Carbon Monoxide Poisoning in Distillation of Coal Tar.

The only recorded accident known to us of *CO poisoning in the Coal Tar Industry* was reported by Greiff[2] in 1890. In fact, till then, the development of this gas in pitch distillation was quite unknown. It was held by the directors of the work in which the accident occurred, that CO poisoning was impossible during the process, but a subsequent inquiry into another death which took place, and the fact that CO was found in the blood of the victim, put the question beyond doubt. In distilling tar, first the light and then the heavy oils are given off, leaving the pitch which forms about 50 per cent. of the tar primarily used. From this pitch cokes are made, and these are often preferred to gas coke as they give practically no ash. The apparatus in this case was a cast-iron retort of 4 cub. metres, with a fire underneath, the whole being surrounded by a wall. In the cover there was a man-hole, also a pipe for carrying away the vapours. A worm apparatus was used for cooling. After heating, the semi-fluid mass is left to cool. In ordinary working the cooling of the retort takes three to four days, when it is emptied by the man-hole. During the process, and owing to the high temperature and dust, the workmen inside are compelled to come out every ten minutes to get fresh air. At the time of the accident, owing to the management having an ample supply of pitch, the cooling of the retort had been allowed to go on for fourteen days instead of the usual three or four. Two men after eight days had taken out a little material, when they were called to other work. After further eight days had elapsed, another workman was sent to finish the work. He was taken out unconscious, and died. The factory doctor certified heat stroke as the cause of death, but the public Prosecutor was not satisfied and ordered a *post-mortem* examination. Light red spots were found on the body, and the blood gave the typical lines of CO-hæmoglobin, even after being kept a year in a bottle.

[1] Memorandum as to Use of Water-Gas, Suction-Gas, and other Gases in Factories. April 1910.

[2] Greiff: *Ueber Kohlenoxydvergiftung bei Theerdestillation. Vierteljahrs. gericht. Med.* 1890 p. 359.

Special experiments were then carried out to find out how the CO was produced. Pieces of the pitch were taken from the middle and sides of the retort and, after examination and analysis, great differences in the physical and chemical composition were discovered. The piece from the side was practically gas-free, but that from the centre of the retort contained in 100 parts about 64 per cent. of hydrogen, 23 per cent. of nitrogen, 2 per cent. of CO_2, traces of CH_4, and 10·7 per cent. of CO. They took 50 c.cm. of this gas and mixed it with fifty times its volume of air. A mouse placed in this mixture died in a few minutes, and its blood showed CO-hæmoglobin. The possibility, then, of generating large quantities of CO at the end of the distillation was proved, and it was supposed that this gas had become occluded in the deeper layers of the coked mass, and further, that when the mass was disturbed by the workmen, the CO gas slowly streamed out of the porous coke.

The reason why the accident occurred on this occasion after the retort had been left to cool for fourteen days, and not when the mass was broken up three or four days after distillation, was probably owing to the fact that when the shorter period had elapsed, the circulation of air in the retort was much greater owing to the considerable difference of temperature between the air outside and that inside. On the day of the accident the weather was warm and sultry, consequently the air in the retort with its large percentage of CO became stagnant, and had sufficient time to act on the man working inside. The management was exonerated from all blame in the matter. To prevent such accidents while the retort is being emptied, there should be a scheme of artificial ventilation by suction.

An unusual case of CO poisoning occurred at a *Lamp-black Work*. To manufacture lamp-black, "the solid residue from tar distillation, a crystalline substance rich in hydrocarbons, is burned on a hearth, with a limited supply of oxygen. A long smoky flame is produced, the products of combustion being carried along a narrow flue, and the lamp-black, in the form of flakes, deposited in a condensing chamber. On the morning of the accident a high wind prevailed, and a spark appears to have been blown along the flue, which set the lamp-black deposit on fire. The substance smouldered for some time before being discovered, and instead of removing all the ventilators in the chamber roof and waiting until fresh air had diluted the atmosphere, deceased opened a man-hole door and entered with a watering-can for the purpose of extinguishing the fire. He was found a few moments afterwards with his face resting among the burning black just inside the entrance. There appears no doubt that the depositing chamber must contain CO in ordinary working, and in all probability the smouldering of the manufactured article would greatly increase the amount present."[1]

[1] *Factory Reports*, 1910, p. 93.

CO Poisoning in the Printing Trade

In the House of Commons, in October 1909, the Secretary of State for the Home Department was asked if his attention had been directed to carbon monoxide poisoning affecting certain workers in the printing trade, which arose from the fumes of gas now being largely used in heating the metal pots attached to linotype composing machines. In reply, the Home Secretary said he had made inquiry into the case and that the reports would appear to show that it was a case of gradual poisoning by carbon monoxide. Poisoning by this gas most usually arose as an accident and had usually a more or less sudden origin. The question of scheduling carbon monoxide poisoning as a disease had been considered by the Industrial Diseases Committee, but they were unable to regard the evidence as sufficient to justify a recommendation of inclusion. Such cases as the above were extremely rare, and no other case of the kind in connection with linotype work was known to the Department.[1]

[1] *B. M. J.*, Vol. II., 1909, 30th Oct.

CHAPTER IX.

SYMPTOMS OF POISONING CAUSED BY BLAST-FURNACE, PRODUCER, AND ILLUMINANT GASES.

Symptoms met with in men suffering from Poisoning by Blast-furnace, Producer, and Illuminant Gases:—

We shall first describe in a general way the signs and symptoms which occur most usually at the time of gassing, as well as those more remote symptoms or after-effects which are so commonly met with, making reference in passing to unusual cases.

With regard to these symptoms it may at the outset be said that they are not of a stereotyped clinical character, for the percentage of CO present in the noxious atmosphere must always be taken into consideration, since upon this will depend the rapidity or slowness of the poisoning and the particular prominence of the symptoms manifested. At the same time, the percentage volume of oxygen present in the respired air must also be observed, because where this is small, poisoning will be much more rapid. It will be noted, in relation to susceptibility, that the age, sex, constitution, and health of the individual, are also by no means unimportant factors.

When poisoning has been slow, definite and distinct stages may be well-marked, but where it has been rapid, these stages are merged into one another. If a man should happen to be working in a confined place where there is a dangerous amount of CO present, he may practically without warning drop unconscious, as if shot, and may succumb very quickly. In many of the cases of poisoning by producer-gas, etc., where the percentage of CO is large, serious symptoms will be found to make their appearance very quickly, constituting what might be called fulminant poisoning by CO, or what the French call "intoxication massive." Happily, however, the gas is not always present in such quantity as to act in this manner.

Many of the symptoms which already have been described under CO poisoning in mines may be met with in men who are working where small quantities of the above gases are present. Among these are giddiness, swimming sensation, ringing in the ears, flashes of light before the eyes, pain or a peculiar sense of constriction in the head, feelings of oppression

in the chest, slight breathlessness which increases progressively, noisy, slow, or irregular breathing, fluttering or throbbing of the heart and of the larger blood-vessels, which beat at first more quickly then more slowly, loss of power in the limbs, especially the lower limbs, followed by absolute muscular helplessness, the patient finally falling to the ground in an unconscious condition. Sir Humphrey Davy, when experimenting on himself with CO, discovered an inability to recognise objects, then a pronounced weakness in the lumbar region, and this passing down into his legs, he dropped unconscious. Some men say that they felt that there was something wrong with them, something which they could not describe or explain; that they worked on in a mechanical sort of way without really knowing that they were doing so, while they had difficulty in judging what was happening.

Another somewhat peculiar symptom which has been described to us by several men who have on more than one occasion been gassed, is that just before the swimming feeling comes on, and just before they begin to collapse, a peculiar sweet taste develops in the mouth. This is regarded by these men as a sure sign of poisoning, and as a danger signal which must on no account be ignored. Witter[1] of Dublin was the first to draw attention to this in his experiments on himself in 1814; but very few others have noted, or at least recorded, its presence. It is also interesting here to note that one of the names given to after-damp is "sweet-damp."

Klebs,[2] experimenting on himself, declared that the first subjective symptom is a burning sensation in the skin of the face, which is quickly followed by giddiness, lights in front of the eyes, beating of the temporal arteries, and headache which gradually increases in intensity. One man who has been gassed on several occasions, describing his sensations to us, said that the feeling of being overcome by gas was exactly like that of being put under chloroform. The unintelligent and stupid who have been gassed cannot generally give one any conception of how they felt, on account, probably, of the insidious nature of the poisoning and the character of the symptoms not being sufficiently insistent. Indeed, in some cases, men fall into a dreamy state in which they may go on working automatically till they drop. Others have an overpowering inclination to sleep, which may soon pass into a state of coma; many cases occur, for example, of persons who have been poisoned by illuminant-gas, etc., during sleep, and of tramps who have lain down to sleep near kilns, etc., or near points where gas is escaping, whose condition seems to be one of passing from sleep to death.

The question as to whether poisoning by charcoal fumes is a painful

[1] Witter: *Eclectic Repertory*, Vol. V., p. 540.,

[2] Klebs: *Virchow's Archiv.*, Bd. XXXII., p. 469.

or painless death gave rise at one time to much discussion in France. Those who believed in the painful death theory founded their view on a few well-known cases, such as that quoted by Faure[1] which is found in nearly all books of French forensic medicine. The following are, briefly, the facts of that case. A young workman named Deal, when all his hopes of success in life seemed to be wrecked, resolved to die by inhaling charcoal fumes, and in order to render a service to science he wrote the following description of his symptoms. We quote the final part of his letter. He lit the charcoal fire at 10.15 p.m.:—"10.30. A very thin cloud of vapour is diffused through the room, the candle appears almost extinguished. I commence to have a violent pain in the head, my eyes are filled with tears; there is general weakness, the pulse is bounding. 10.40. My candle is out. My lamp still burns; my temples are beating as if the veins were going to burst. I have an inclination to sleep. I have terrible pain in the stomach. 10.50. I feel suffocated, strange ideas present themselves to my mind; it is with difficulty I can breathe. I cannot go far. I have symptoms of madness. 10.60. I can hardly write any longer; my sight troubles me; my lamp is out; surely I have not much more to suffer before I die. 10.62—" (There are only some illegible characters.) The exaggerated and emotional character of this letter may be compared with that of the letter of farewell to his wife and children written by Sir C. Le Neve Foster at the Snaefell disaster (see page 126). Boehm[2] also writes:—"The psychical condition which precedes loss of consciousness is sometimes a distress of mind full of agonising tortures; sometimes a sort of pleasant and ecstatic feeling; but the former appears to be the rule. The access of insensibility is either a sudden withdrawal from consciousness, like a stroke, or it is preceded by pronounced phenomena of great discomfort, anxiety, and excitement, which often lead the poisoned persons to make an effort to leave the poisonous atmosphere in which they find themselves, or to try to get air by opening the windows, etc."

We have given the foregoing in detail as it sums up very accurately the views of some writers who believe that asphyxiation by CO is a painful process. This, however, is quite contrary to the experience of most writers, who hold that illuminant and producer gases, charcoal fumes, etc., have rather a benumbing or soporific effect. Doctors who have themselves experienced poisoning by CO accidentally, or while carrying out experiments, hold that the effect, after the preliminary discomfort passes off, is not painful. We know that in many cases coal-gas has a curious dulling effect on the brain, for patients have been severely hurt and burned without feeling any pain. It is interesting to remember in

[1] Faure: *Archives Générales*, 1856, p. 39.
[2] Boehm: *Ziemssen's Cyclopoedia of Med.*, Vol. XVII., p. 465.

this connection that Sir James Simpson was well aware of this peculiar anæsthetic influence of coal-gas, and thought at one time of using it as a substitute for chloroform, but the variability of its action and the difficulty in estimating it prevented him doing so. Indeed it is this benumbing action on the brain which makes CO poisoning so insidious, as the patient often does not perceive that anything unusual is happening till poisoning is well advanced, and when he does so it is perhaps too late.

A remarkable case reported by Faure depicts graphically the insidiousness of its toxic action. A woman was found dying in bed. A number of people gathered around her. All of these left for a little time except one who began to feel ill, complaining of headache and noises in the ears. This woman fought against this condition, which she could not explain, then she fainted; another woman who helped the first two victims also collapsed, then a fourth. They were carried to another room, and there they quickly recovered. Some of them returned and were again seized by the same symptoms. Others who had been assisting in the intervals had been successively overcome. On the arrival of the doctor all the patients were taken out of the room. Many hours afterwards it was found that wood planks placed deeply in the kitchen wall near the fireplace had caught fire, and the gases of combustion had found their way into the room by slits and crevices concealed in a cupboard. One can readily understand had these cases occurred at night, that death could quite easily have resulted without the people exposed to the gases being awakened. The insidious action of blast-furnace and producer-gas cannot be too strongly emphasised, as in many instances men are rendered unconscious without practically any premonitory warning. Nearly all who have experienced the effects of poisoning by CO are agreed that pain is not a dominant condition in the process.

While, however, it happens that most persons receive little warning from the onset of symptoms regarding the dangerous atmosphere in which they are working, or are placed, some are able themselves to perceive danger and at the same time to warn their neighbours. Again, some will declare that they felt and understood perfectly that they were being gassed and in danger, but that owing to the development of that utter powerlessness in the lower limbs, which is such a marked characteristic of CO poisoning, they were unable to drag themselves away. Dramatic pictures of this are seen after large colliery explosions and after fires. Brouardel,[1] in his report on the fire at the Opera Comique, for example, describes how in a refreshment room twenty-seven persons there met their death, whose bodies showed no signs of being burned, their very combustible clothing being untouched, and yet none of whom had made any attempt to escape.

[1] Brouardel: *Les Asphyxies par les Gaz, etc.* Paris, 1896, pp. 28 and 77.

A marked example of the powerlessness of the limbs, along with perfect consciousness and retention of reasoning faculties, is furnished by *Briand.*[1] A woman, afflicted by insanity, believing herself persecuted by her neighbours, made up her mind to die, together with her husband (who was weak-minded), and her daughter, a girl of sixteen years. After considerable difficulty she persuaded her husband to agree with her view. Accordingly, having lit the stove and seen that her husband and daughter were lying down, she got into bed beside them, encouraging them all the while with promises of everlasting happiness. The father who was nearest the stove was the first to die, and shortly afterwards the mother also succumbed. All this took place on a Sunday evening; and on the Wednesday morning on the room being entered, the girl was found lying between the two corpses. She retained a perfectly clear recollection of all that had passed. She said:—"I wiped my mother's mouth while she was in agony, but it was impossible for me to rise; my legs were paralysed." When the girl found that her parents were quite dead and that no one replied to her cries for help, she went to sleep fully expecting to die, but, instead, woke up with paralysis of both legs. In this case the patient had a complete recollection of all that had passed, up till and even after the death of her parents. We have already drawn attention to one or two cases where this powerlessness of the limbs prevented the person giving the alarm, involving that person in serious legal consequences (see p. 5).

Effect of CO poisoning on Animals.

It will be interesting and instructive to consider shortly the symptoms of poisoning in animals. The following account is gathered from the experimental work of a number of observers. In all these experiments carried out on animals, it is remarkable that the after-effects and sequelæ found so frequently in man do not appear in animals. The guinea-pig, for example, shows signs of disturbance; then it commences to rub its snout as if something were irritating it; breathing becomes more rapid, but after a short time much less rapid; the blood-vessels of the ear appear to enlarge (Klebs);[2] then the animal's hind legs give way and it falls back, then on its side; convulsions soon appear, and the animal passes into a comatose condition; the temperature of the body, which from the beginning of poisoning has been gradually getting less, now drops more rapidly, and the body becomes very cold; the pulse can hardly be felt; and the breathing is slow and interrupted by frequent pauses.

[1] Briand: *Annales d'Hygiène Publique, etc.*, 1889, Vol. XXI., p. 362.
[2] Klebs: *Virchow's Archiv.*, Bd. XXXII., p. 4.

In man, while in the unconscious condition, we may find certain motor disturbances ranging from simple restlessness, shivering, trembling, marked muscular tremor, rigidity and spasm of certain muscles or groups of muscles and trismus, to the most violent tonic and clonic convulsions.

In every case where the poisoning has been severe, there is profound disturbance of the sensibility. It is often found that there is complete anæsthesia of the skin of the whole body; but Faure, in his careful study of asphyxia, pointed out that although there might be profound anæsthesia in all other parts of the body, yet the upper part of the chest might remain sensitive; and he showed how a knowledge of this might be utilised in treatment by provoking reflex respiratory movement by the application of heat. As this part of the body is the last to lose sensation, so is it the first to recover. This anæsthesia may be distributed in patches over the body. It generally disappears very quickly after the patient recovers consciousness, but in other cases it may last for a considerable time. Or again, a remarkable hyperæsthesia of the skin may develop; the slightest touch being sufficient to produce severe muscular spasms and convulsions.

Localised swellings in various parts of the body have also been found in the acute stage of poisoning. We have already pointed out that Klebs (while other German observers as Eulenberg, Biefel, and Poleck have also described this in their experiments) was the first to point out that in an early stage of poisoning there was intense congestion of the ears, accompanied by a peculiar trembling of the body.

When discussing the sequelæ of CO poisoning, it will be noted very early under exposure to the gas, that bright cherry-red hyperæmic patches may be found in different parts of the body. For example, in the case of Leudet described at length later, there was a bright red patch over the area of distribution of the musculo-spiral nerve, and another on the right temple. In a case of Munzer and Palma,[1] large patches (2 in. sq.), of a whitish colour with infiltration of tissue underneath appeared, one on the right zygomatic region, and a similar patch on the right side of the forehead. In the same case the skin over the thorax was very red, and on the inner side of the right forearm a broad red swelling (3 by 2 in.) showed itself. There were similar red patches on both knees. These changes in the skin persisted for three days. More falls to be said about the motor, sensory, and vaso-motor nerve disturbances when discussing the sequelæ of CO poisoning.

Munzer and Palma: *Zeitschrift für Heilkunde*, 1894, Vol. XV., p. 186.

Symptoms which point to urgent danger, or to the case having a fatal termination—Prognosis:—

As the case becomes worse, the pulse-rate becomes much diminished, falling perhaps as low as 35 or 45 per minute, and at the same time respiration becomes slow and superficial, or laborious and irregular, and there are frequent pauses; the coma also becomes deeper. As the symptoms progressively advance, the pulse-rate becomes again much increased, the pulse-beat very weak, indeed hardly perceptible, and at the same time the surface of the body becomes very cold, as if dead. In other cases the heart progressively beats more and more slowly, then after a few precipitate beats there comes a long pause, then rapid, weak action till death occurs. In some cases the patient has the appearance of having had an apoplectic seizure, with noisy stertorous breathing which is often jerky and irregular, puffiing of the cheeks, perhaps great pallor of the face with lividity of the lips, while in other cases there are cyanosis of the face, widely dilated pupils not reacting to light, deep coma, and complete paralysis. The patient may be in a comatose condition for days and then die; in such cases the end is often sudden. Where there is prolonged coma, the prognosis is nearly always grave, although cases have recovered completely where the patient had been unconscious for a number of days. Generally, however, when these cases recover, the victims suffer for a considerable time, in some cases permanently, from various mental and nervous affections.

In considering prognosis in all cases where nerve lesions have developed, we have to decide whether these are peripheral or central, and if the latter, whether or not they are due to gross lesions in the brain. Such conditions may develop several days or even weeks after the patient has apparently recovered from the immediate effects of the gas poisoning. Many of these lesions do not prove very amenable to treatment. We generally find, however, that where the lesion is peripheral, there is complete recovery perhaps after a lengthened illness. Where the symptoms are produced by gross lesions, such as extensive cerebral hæmorrhages and softenings such as have been described by Poelchen, Simon, and others, death nearly always takes place.

In some fatal cases of charcoal, illuminant, and producer-gas poisoning, it has been observed that the pupils were contracted almost to a pin-point size, suggesting opium poisoning or hæmorrhage into the pons Varolii; or, again, the pupils have been found alternately widely dilated and contracted within short intervals.

The patient may die in a convulsive seizure. This phenomenon is much more commonly met with in producer and illuminant-gas poisoning than in gas poisoning in collieries. In some fatal cases the patient has been found comatose, and the body in a semi-rigid condition.

It must be enforced on the attention that in cases of gas poisoning which seem to be slowly improving, death may take place very suddenly, although up to that time nothing pointed to a fatal termination. In many other cases the symptoms change from bad to worse, and *vice versa*, in a very extraordinary manner. A good description is given by Taylor[1] of this peculiar and characteristic fluctuation of symptoms in CO poisoning. He writes: "These were alternately encouraging and discouraging, the system at one time striving vigorously to work off the poisonous load and anon sinking exhausted with the effort. Thus the periods of hopeful tranquillity, when the pulse was steady and the breathing regular, were always followed by intervals of shorter duration, when all these functions were deranged." Golding Bird,[2] in writing of the deceptive character of CO poisoning and its symptoms, says that cases "partially revive in fresh air exciting hopes too frequently fallacious, as certain cases almost always die, even after living for several days in a state of somnolency."

Indeed in all serious cases it is found that relapses are frequent even after the patient has considerably improved and been conscious for a considerable time, and many observers quote cases where the patient was brought round again and again only to fall back into a stupor. With regard to prognosis from the length of the period of exposure and the percentage of CO present in the atmosphere, we repeat what has been previously said, that the longer the exposure even to small quantities of CO and the higher the percentage of CO present in other cases, the more likely do serious consequences, even death, result, and the more likely are serious after-effects to develop.

Sudden death after poisoning by Carbon Monoxide.

Reference has already been made under poisoning by after-damp to the fact that sudden death is by no means uncommon in persons who have been exposed to CO. The fact that death often occurs suddenly and unexpectedly after poisoning by charcoal fumes has been long recognised. Faure[3], writing in 1856, lays great stress on this, and quotes the following case. A man, with his child who died later, was asphyxiated. When found, the father was very ill. He quickly recovered, however, in the hospital, was able to walk about and carry on intelligent conversation, but during the evening he suddenly expired without any new symptom developing.

The following are a few typical cases occurring after producer and illuminant-gas poisoning.

[1] Wm. Taylor: *Edin. Med. Jour.*, 1874, Vol. XX., p. 20.
[2] Golding Bird: *Guy's Hospital Reports*, Vol. IV., p. 75.
[3] Faure: *Archives Générales de Méd.*, 1856, p. 44.

A workman, in perfect health, who was employed on the gas-plant in connection with a Bleach-works, had occasion to go above a purifying box to remove the cover. He came down complaining of being ill, suddenly became unconscious, and died in a few minutes. It was shown that death was due to carbonic oxide poisoning.[1]

An engineman, who was in charge of an engine through which air and water-gas were pumped for welding purposes, was overcome at his work, but *was able to walk home.* He died very suddenly three hours afterwards. The jury returned a verdict of failure of the heart, accelerated by water-gas poisoning. At an Ironworks near Newcastle, a few years ago, a man died very suddenly after breathing for only a few minutes air heavily charged with CO.

In connection with such cases, it is well to repeat the warning of the late Dr Dixon Mann,[2] that if there is a large percentage of CO present in the atmosphere, death may occur before the hæmoglobin becomes sufficiently charged with it to afford spectroscopic evidence of any change. In 1903, Garnier[3] reported the case of a workman who was looking after some fittings about a Gaswork and who was overcome by gas. When brought round, he recovered sufficiently to enter into conversation with his wife. He complained of vertigo, epigastric pain and oppression, and indefinable weakness, and suddenly died in a quarter of an hour. Dr W. Taylor,[4] in calling attention to the not infrequent sudden death after coal-gas poisoning, mentions the case of a workman, who, stooping down to examine a gas main, which had been broken and was leaking badly, inhaled a very large quantity of gas and immediately dropped down dead. Casper[5] also reports a case where a man, twenty years of age, entered a room full of illuminant-gas, and after a few inhalations dropped down dead.

When CO has been inhaled for a considerable time, the damage done to the nervous tissues, especially the vital nerve centres, may be very serious owing to the long deprivation of the body of oxygen. It is very important to remember the peculiar action of CO on the nervous mechanism of the heart. Many cases are on record of men who had been overcome by gas and rendered unconscious, and who in time were supposed to have recovered, suddenly dying.

What has been said above will probably also explain those instances in which men, from the nature of their occupations, were exposed to gas poisoning for long periods. Although these men were never really over-

[1] *Factory Reports*, 1900, p. 497.

[2] Dixon Mann: *Forensic Medicine and Toxicology.*

[3] M. L. Garnier: *Comptes Rendus de la Société de Biologie*, 1903, p. 761.

[4] Wm. Taylor: *Edin. Med. Journ.*, 1874, p. 273.

[5] Casper: *Archiv. für Psych.* I., p. 273.

come by the gas, nor were rendered unconscious, yet they died afterwards very suddenly. The punctiform hæmorrhages and other lesions in the brain described by Cramer, Schaefer, Mott, and others, not only explain many of the remote effects complained of after CO poisoning, but also many of the cases of sudden death. Artigalas[1] calls attention to sudden death occurring 24, 36, and 48 hours after accidental gassing, when all acute danger appeared to have passed, and to the fact that in these cases the breathing suddenly ceased. This he regarded as due to some bulbar lesion. Because of the fact that diabetes is found in CO poisoning, and that this can be produced experimentally by puncture of the floor of the fourth ventricle, and because the vagus nerve root situated in this region is frequently affected in CO poisoning, he sees nothing surprising in the occurrence of sudden death; the arrest of the respiration, with momentary increase of the heart beat, in these temporary but false recoveries, being all accountable to lesions of the bulb caused by the antecedent toxic condition.

When a patient who has been gassed regains consciousness, he has generally a stupid, dazed look. Although many will be able to tell what occurred immediately before they were overcome, where and what they were working at, and how they felt, others have completely forgotten everything immediately before they became unconscious, that period seemingly being wholly erased from their minds. With the dull, heavy, dazed look there is often slowness and defect of speech, the patient appears to think slowly, giving an appearance of being drunk with alcohol, and the difficulty which he has in comprehending what is said to him adds to this effect. Others, again, may have rather a peculiar look: their faces are white, and they have a wild, frightened appearance, a large part of the white of the eye being shown, thus producing a startled, staring appearance. Among women who have been gassed by charcoal fumes, it is often noticed that they are very excited and frightened when they recover consciousness, as though they were in dread of something happening, staring round them in a wild, startled way. Golding Bird[2] pointed out that some patients immediately after recovery are seized with an indefinable, vague feeling of horror or dread, which is rapidly succeeded by an irresistible disposition to somnolency or syncope. The patient, immediately after recovering, and perhaps for days afterwards, often complains of a feeling of intense and general weakness and of the most extreme fatigue, with aching pains in many of the muscles of the body. For a few hours after recovery the patient may yawn incessantly.

The colour and appearance of the face depends a good deal upon the

[1] Artigalas: "Des Asphyxies Toxiques," *Thèse de Paris*, p. 71.

[2] Golding Bird: *Guy's Hospital Reports*, Vol. IV., p. 85.

rapidity of asphyxiation, the amount of CO in the air, and also upon individual peculiarities, for we find that this may vary widely in different persons exposed to the poisonous gas under the same conditions and for the same time. The countenance may be swollen, dusky-coloured, and bloated, or pale and bedewed with perspiration, but in some cases where asphyxiation has been rapid it has a leaden tinge; whereas in others it may be of a brilliant, strikingly red colour, the lips being like carmine, and the cheeks pink. In some fatal cases the face preserves the appearance of life to such an extent that the friends of the deceased, four or five days after the death, will hardly believe that the patient is dead. Stevenson, Brouardel, and several others mention such cases, and Firmin Dervieux reports a few among the victims of the Courrières disaster. The eyes are sometimes found much injected and congested, although in other cases the opposite is found, the sclerotic coat being a brilliant white. In some cases a peculiar rhythmical oscillation of the eye-balls has been noted.

When the comatose condition has been deep and prolonged, it may be found that thick mucus, perhaps tinged with blood, flows from the mouth and nose, and the noisy, rattling breathing proclaims that a considerable amount of mucus is present in the bronchial tubes. This is probably the result of the œdema of the lungs produced by the stasis of the circulation.

Many men, immediately on recovering consciousness after poisoning by furnace or producer-gas, behave as if they were intoxicated by alcohol. This may occur even in the case of the steadiest and most sober of the workmen, although mental symptoms are much more marked among drinkers. The patient may stagger about, waving his arms in an erratic manner, and talking or shouting incoherently. A few become very quarrelsome and pugnacious, and there is great difficulty in restraining them. Casper-Liman[1] reports the case of a shipowner, twenty-four years of age, who was poisoned by CO and in whom a remarkable state of excitement appeared as soon as he got into the open air. On going immediately afterwards into a drinking bar his mind suddenly became confused, he declared he was the devil, became very aggressive, almost maniacal, quarrelled with everybody round about him, smashed chairs, etc., and fought with the policeman who was called to arrest him. After a deep sleep, he woke up perfectly clear in mind, having no recollection of anything that had happened. Jergolsky[2] also reported a case in which furious delirium developed after poisoning by charcoal vapours. This lasted for seven hours, and disappeared without leaving

[1] Casper-Liman: *Handbuch der gericht Med.* Berlin, 1889, Vol. II., p. 579.

[2] Jergolsky: *Bull. des Séances de la Soc. Méd. de Kalouga.* Russia, 1897, p. 47.

a trace behind. He believed that these cases of passing delirium must be very much more common than the number of cases reported in medical literature would lead one to believe. Indeed, owing to their fleeting character, the doctor may not see them. Other workmen, poisoned by furnace and producer-gas, develop a hysterical condition, weeping or laughing alternately, or they may become very restless and excited, and sometimes they burst into song. Since *Devergie*[1] called attention to the fact that, when the patient comes to his senses, furious and maniacal delirium may set in from 36 hours to 5 days after gas poisoning, a number of cases have been reported by Bourdon, Thomson, and others, to which we shall call attention (p. 282).

Briand[2] describes a patient, a young girl of eighteen, who, when attempting suicide by charcoal poisoning, severely burned her left arm to the third degree. When found and roused up by the neighbours, she appeared confused, and, at the same time, much surprised at the appearance of so many people around her. She did not appear to notice her severe burn, and, becoming very excited, began to recite, then sang several songs, in the intervals of which she called for her grandmother who had been dead six months, gesticulating all the time in a wild manner, violently waving about her injured arm.

Sometimes men get what they call a dose of "laughing gas," so called from the effect which the gas has on the patient. The gas in these cases is essentially the same, and there is absolutely no trace whatever of nitrous oxide present. Here the man dances or staggers about, waving his arms, laughs or even roars with laughter whenever he is questioned or spoken to, while attacks of immoderate laughter may come on spontaneously. In the case of a furnace-man who had been poisoned by furnace-gas, and in whom there was nystagmus, incomplete loss of power of limbs with development of steppage gait, and well-marked mental symptoms, Sir Thos. Oliver[3] found, two months after the accident, that the patient would burst out into hilarious laughter now and again without the least provocation. The simplest question addressed to him seemed to cause amusement. Croizet[4] reports the case of a sea captain so seriously poisoned that both arms and legs were completely paralysed, who was quite incoherent, and who talked in a most excitable manner. In a loud voice he went through the leading actions of his sea-life, giving orders, etc., every now and then being attacked by fits of weeping.

[1] Devergie: *Méd. legale.*, 1852.
[2] Briand: *Annales d'Hygiène Publique, etc.*, 1889, Vol. XXI., p. 360.
[3] Thomas Oliver: *Diseases of Occupation*, 1908, p. 60.
[4] Croizet: *Thèse de Paris*, 1903, p. 55.

After-symptoms.

Headache is one of the first symptoms to appear and often the last to disappear. Even when the poisoning has been very slight, the patient generally complains in a short time of severe headache. When a man starts work in a place where there is a small amount of gas present, for example in a gas-engine house, he generally complains of headache until toleration becomes established. This headache often does not appear, however, until the person affected goes out into the fresh air.

After gas-poisoning many complain strongly of pain in the head. This is very often of a dull aching character, either frontal, or over the top of the head, or it may be present as a throbbing pain in the temples, or a sense of "a painful constriction of the temples" (Brouardel)[1]. This sense of constriction in the head was first pointed out in the contribution on Asphyxia by Faure,[2] who says that in the case of sixty miners poisoned by fumes from burning wood in a mine, it was complained of by a large number. He holds that the first symptom complained of is this constriction with headache. Golding Bird,[3] who was the first British writer to make a thorough clinical study of charcoal and coal-gas poisoning, describes this symptom as an "intense pain in the head with a sense of constriction round the temples and forehead resembling that produced by a tightly bound cord." We have sometimes seen men who were recovering from gas-poisoning sitting on the ground holding and pressing their heads between their hands, and who declared that their heads felt as if they would burst open. When the headache is very severe, the pain itself may bring on attacks of nausea and vomiting. Some will affirm that accompanying the pain of a peculiar penetrating kind, is a sense of weight and heat, more especially in the occipital region; the head feels too heavy. One man whom we attended complained of the pain being localised at a point, and that it felt "like something being driven into his head by slow degrees." Chevalier [4] tells of a case where the patient described her symptoms as "if a hammer were going at it in her head." This patient continued to have neuralgic headaches for months afterwards. Vergely had a patient who complained of *a very painful sensation of heat* in the head, which was soon transformed into headache. In men who have been poisoned by furnace-gas, we have observed a peculiar sensitiveness of the scalp which lasted a couple of days; slight contact with the hair, without pressing the scalp, causing considerable pain. In every case the headache is usually intensified by

[1] Brouardel: *Les Asphyxies par les Gaz., etc.*, Paris, 1897, p. 27.
[2] Faure: *Archives Générales de Méd.*, 1856, p. 35.
[3] Golding Bird: *Guy's Hospital Reports*, Vol. VI., p. 83.
[4] A. Chevalier: *Annales d'Hygiène Publique*, Vol. XXII. 1864, p. 73.

muscular movement. In exceptional cases there may be no headache, but in such cases there is sometimes overwhelming giddiness. The headache generally disappears in a few days; but in some cases it lasts much longer, and in others, as that described by Chevalier, it may recur at regular intervals for months after poisoning.

In some cases the most prominent symptom is *pain in the back* and shooting pains down the legs; while many patients complain severely of vague indefinable pains for weeks after their exposure to gas. When Sir Humphry Davy [1] recovered consciousness, after having been overcome by CO while carrying out his experiments, he suffered from giddiness, sickness, headache, extreme debility, and acute pains in different parts of the body. These pains are probably of a neuralgic character. We shall refer to them again when discussing neuritis.

Thoracic pain.—In some cases the patient complains of violent pain of a peculiar rending or tearing character in the region of the sternum. The following case in which this was a marked feature, is especially interesting because of the fact that it occurred after the first experiments carried out with furnace-gas, and also because of the author's extraordinary explanation of the causation of the pain. In 1846, Adrien Chenot, [2] the chemist, being anxious to ascertain the properties of the gases yielded by smelting zinc ores with charcoal, inhaled them directly into his lungs through a pipette. After a few inhalations he fell as if stunned, his eyes were turned up, his skin became discoloured his veins swollen, he had most violent pains in the chest, and his brain felt powerfully oppressed. Then sensibility appeared remarkably intensified—"life, so to speak, was exalted, and the most vivid dreams were reproduced as if in an instantaneous mirage." Coming more to himself he complained of dreadful thoracic pain, as if his internal organs were being torn asunder. For several days afterwards he complained of the severity of these pains, felt depressed and languid, sleep was heavy but frequently disturbed by the pain of cramp in the knees and toes, and for months afterwards there was a morbidly excited state of the nervous system. He thought that the CO was oxidised in the blood to CO_2, and that the abstraction of the oxygen of the blood, and its action on the carbon produced a considerable quantity of heat. He believed that the cause of the great pain inside the chest was this sudden elevation of the temperature.

Vertigo.—During exposure, headache and giddiness are the first symptoms to be complained of, and on recovery the first and most commonly felt. Vertigo is often marked, and it generally persists for a

[1] Davy: *Elements of Chemical Philosophy*, p. 172.
[2] Chenot: *Gazette Médicale*, 17th Apr., 1854.

short time only, usually not longer than one or two days, although in some cases, as in that of Dr Motet,[1] it may persist for several weeks. Occasionally the vertigo is the chief symptom complained of, although generally when it is present there are, in addition, headache, nausea, and vomiting. In these cases the vertigo is frequently so violent that the patient cannot walk or even stand. Vertigo may be accompanied by noises in the ears. It often disappears immediately the patient lies down, which points to it being caused by circulatory disturbances in the brain. We have frequently drawn attention to the fact that in CO poisoning we are dealing with oxygen-starvation of the tissues, and it is noteworthy that like symptoms are very prominent in those suffering from mountain sickness, and in the anæmias and after-hæmorrhages where there is similar oxygen-starvation.

In some cases the patient, even when lying down, is not free from giddiness till he closes his eyes. Whenever he opens them the vertigo returns, and with its return there is often severe nausea. In other cases the feeling of swimming appears as if outside the person; that is, when looking at objects they appear to swim or whirl about him in circles; or the movements may appear more rapid and precipitate, objects appearing as if they were being dashed about. Sometimes the movements of objects looked at are rhythmical, moving from side to side or up and down at regular intervals. Dr Motet complained very much of these illusions of sight. While lying in bed and looking at the gasalier before him it suddenly passed to the right, just as, he says, an object might do when looked at from the window of an express train. This illusion was mixed up with another, viz. :—two pictures near each other on the same wall began to rise and fall at regular intervals. He had the idea that these movements were synchronous with the beats of the heart. In most cases, as in the foregoing, the patient is perfectly aware that these movements are illusions, but in other patients the whole mind appears to have become confused, and they are apt to think that the movements are actually taking place. In cases in which deafness resulted, as in Bourdon's cases, vertigo was very pronounced.

Sweating.—In some cases when the patient is recovering, he has attacks of copious sweating. This is one of the vaso-motor disturbances which later we shall have occasion to note.

Vomiting.—During exposure to CO vomiting may take place. It is very frequently found after the patient partly recovers, the vomitus being often composed of bilious matter. At the Crarae disaster two or three of the survivors vomited matter like coffee-grounds, and this symptom has been noticed by other observers in other instances. Bloch had a case of CO poisoning where the patient vomited considerable quantities

[1] Motet: *Annales d'Hygiène Publique*, Vol. XXXI., 1894, p. 262.

of blood for three days. We have also heard men complaining of severe *epigastric pain*, which is relieved, however, by the vomiting which generally follows in these cases. It may have been the relief of pain obtained by vomiting which prompted the practice so frequently found in ironworks of administering an emetic in all cases of "gassing." In some cases there has been found marked distension of the abdomen, which generally disappeared, however, when an enema of turpentine, soap, and water was administered. In others there is retraction of the abdomen, as in cases reported by Thomson,[1] Simon,[2] and others.

Diarrhœa.—We have occasionally seen cases of men who, when they had a "feed of gas" as they put it, instead of having vomiting with perhaps epigastric pain, had severe attacks of diarrhœa with colic, but which generally passed off in a day or so. One of these men had been gassed on several occasions, and on each he had severe diarrhœa with colic which lasted for a couple of days, accompanied by the usual headache and giddiness. This symptom we have also noted in a number of cases of black-damp poisoning; indeed, we believe that it is very common in this form of gas-poisoning; and numerous cases are recorded of it in poisoning by illuminant-gas, when it was a prominent symptom. *Biggam*[3] found, for example, that out of seven persons who were exposed at the same time to CO poisoning, and of whom two died, in two of the others diarrhœa was a marked symptom. Although comparatively little attention has been paid by writers to this symptom, we should say that it is fairly common, although certainly not so frequent as vomiting. In some cases there is blood in the motions. *Greidenberg*[4] mentions a case where very severe diarrhœa, almost like dysentery, was a prominent feature of a case of CO poisoning.

Relaxation of the Sphincters.—In a case which we attended and which ended fatally, the man never regaining consciousness, there was relaxation of the sphincters with involuntary passage of urine and fæces. We have been told that in India, where cases of charcoal poisoning are very common among the natives, that when they are found in the morning, signs of involuntary movement of the bowels are nearly always present. Devergie was one of the first to point out that this was fairly common. Brouardel, on the other hand, thinks that it is quite exceptional. From a careful perusal of the literature of this subject, we have come to the conclusion that it is much commoner than Brouardel[5] believed, and that in serious cases of poisoning, where the percentage of CO in the air is

[1] Thomson: *Lancet*, 6th May 1903.
[2] Simon: *Archiv. für Psychiatrie*, Bd. I., 1868-69.
[3] Biggam: "Poisoning by Carbonic Acid (CO?)," *B.M.J.*, 28th Jan. 1893.
[4] Greidenberg: *Ann. Méd. Psych.*, July-August, 1900, p. 65.
[5] Brouardel: *Les Asphyxies par les Gaz. etc.*, Paris, 1897.

high, and where poisoning has been rapid, it may be met with frequently.

In fact, wherever the coma has been profound and prolonged it may be found, since it is not an unusual symptom in asphyxiation from any cause. In some cases paralysis of the sphincters may be a prominent symptom and may last for a considerable time. *Broadbent*[1] records a case where, in a man who had been gassed, there were involuntary motions, and in whom this condition existed from the first day and continued until nineteen days later, when he died. Monod[2] reported the case of a woman, unconscious for three days after CO poisoning, who had paralysis of the bladder which lasted fourteen days before recovery; and Laroche,[3] of a woman of fifty-seven who had attempted suicide by inhaling fumes from a stove, and who had paralysis of the bladder for more than a week when she was taken to the hospital; electrical treatment, however, soon bringing about recovery. In a case by Oppolzer,[4] paralysis of the rectum and bladder lasted three months.

Bronchial Symptoms.—Lancereaux,[5] in an exhaustive article on charcoal fumes poisoning, lays considerable stress on the development of a *cough*,—an irritating, short, hard, guttural, tracheal cough, which, he declares, is very frequently found. In poisoning by furnace and producer-gas which has not been washed and cleaned, this cough may sometimes be observed, and we have met with two cases of CO poisoning of men in mines, in whom it was a painful symptom. In one of these the violent coughing led to a brisk hæmopytsis. In both cases the patients complained at the same time of choking sensations in the throat, expressed by them as a "grip at the throat."

Hæmoptysis.—In several cases which have come under our notice after furnace-gas poisoning, the patient, when he had sufficiently recovered to walk home, had slight attacks of hæmoptysis. Several cases are on record where severe attacks of this occurred after exposure to after-damp succeeding explosions, and we had one patient who, three days after he had been poisoned by CO from an underground fire, had a brisk hæmoptysis which lasted four days. A fatal case, that of a man in charge of a suction-gas plant, is recorded in one of the Factory Reports. In this case there was general weakness, nervousness, headache, and nausea, which terminated in a severe hæmorrhage from the lungs of bright cherry-red blood.

Epistaxis may also be met with, and in some cases may be very

[1] Broadbent: *Brit. Med. Journal*, 1893.
[2] Monod: *Gazette des Hôpitaux.* Feb., 1854.
[3] Laroche: *Thèse de Paris*, 1865.
[4] Oppolzer: *Wien. Med. Wochenschr*, 1865, pp. 10, 11.
[5] Lancereaux: *Bulletin de l'Academie de Méd.*, 1889, Vol. XXI.-XXII., p. 161.

marked, as in one which had a fatal termination.[1] Bloch [2] reports the following remarkable case of bleeding from the nose, throat, mouth, and lungs. A fireman, 36 years of age, was poisoned by CO in 1895. When resuscitated, he coughed up blood and had severe bleeding from the nose. He was ill from six to eight weeks, during which time he had severe headache, and felt as if he were drunk. Nearly every day during this time he had bleedings from the nose which were very difficult to stop. From 1895 to 1899 he had recurring attacks of this bleeding, and attended hospital as an out-door patient. Since his accident *he lost the power of smell.* His skin was rich in pigment, but in some places this was absent. This *vitiligo* developed after the accident in 1895 and increased considerably in extent thereafter. He occasionally had severe attacks of palpitation of the heart. In July 1900, he had a severe attack of this, with giddiness and bleeding from the throat and nose. His face was reddish in colour, with dilated vessels. On the lips and throat pin-head sized petechial spots could be seen. In addition to these fresh dot-like hæmorrhages, there were numerous dilated vessels in the mucous membranes of the throat, nose, and conjunctivæ. On exertion there was oppression of the chest, palpitation of the heart, sweating of the palms, and tremor of the hands. Bloch considered this case typical of CO poisoning, from the tropho-neurosis producing vitiligo, the atony of the middle coat of the blood-vessels resulting in inclination to bleeding, dilatation of vessels, and increased blood pressure in the head, and from the direct injury to special nerves, to the olfactory in producing loss of smell, and to the vagus in causing palpitation of the heart.

Hiccough is sometimes met with, is often very irritating and distressing, and is sometimes most intractable to treatment. We have already described a remarkable case of this in one of the survivors of the Courrières disaster (p. 119).

Effect of Carbon Monoxide Poisoning on Body Temperature.

In examining patients who are in a comatose condition from CO poisoning, all observers are struck by the coldness of the body surface; even in minor cases of poisoning there is considerable fall in temperature, the extremities generally being icy cold and stiff as if dead. Pokrowsky[3] made a searching investigation respecting the effect of CO on body temperature. In his experiments each of the animals showed a constant lowering in the temperature, varying in extent from 0·37° to

[1] Parker. *Medical Gazette*, Vol. XLVII., p. 412.

[2] Bloch. *Inaug. Dissert.* Leipzig, 1902.

[3] Pokrowsky: *Virchow's Archiv.*, Vol. XXX., p. 536.

2·7°C. Other observers have found the depression from the normal to be as much as 3°C. Pokrowsky and Klebs[1] affirm that this declension only went a certain length and then stopped. In all Pokrowsky's experiments the percentage of CO present in the gas exhibited was very high, and poisoning was very acute. Borzyszkowski[2] carried out a large number of similar experiments. He found that at the beginning of an experiment there was a slight increase in the temperature, which, however, was followed quickly by a greater fall. The increased temperature, he thought, was due to the increased activity of the vaso-motor centres.

When the patient recovers consciousness, he generally complains of shivering with cold and of inability to keep warm. These attacks of shivering may be very severe, just like a rigor, and in some cases are so exaggerated as almost to simulate a convulsion. For a few days the patient may hang over the fire, continually complaining of the cold, but this condition generally passes off very quickly. In many cases the patient has alternate attacks of shivering and flushings of heat which indicate vaso-motor disturbances. More will be said of this when dealing with nervous disorders. A patient of Wolff,[3] who quickly recovered from poisoning, had an attack of shivering the following day, which lasted fifteen minutes, and this was followed by profuse sweating which ushered in severe convulsions lasting for some time. Regarding the action of CO in poisoning by nickel carbonyl, M'Kendrick and Snodgrass,[4] struck by the fact that by giving to animals small graduated doses of $Ni(CO)_4$ (in which they regarded the poisonous agent as CO), they could reduce the body temperature, thought that that compound might have valuable antipyretic properties.

As regards the causation of this fall in temperature, Chenot and Pokrowsky held that it was the result of defective oxidation. Borzyszkowski believed that it was due to the final atony of the vessels of the skin and the greater loss of heat, as well as to diminution of the oxidation processes owing to so many of the red blood corpuscles being thrown out of action. We believe that the fall in the body temperature in CO poisoning may be brought about in two ways; first, by the combination of CO with the hæmoglobin, the tissues being deprived of a considerable amount of usable blood, and that this is aggravated by the indirect action of the weakened circulation owing to the changed blood on the nervous mechanism of the heart, producing in the end decreased oxygenation with decreased production of heat; and second, by the heat-

[1] Klebs: *Virchow's Archiv.*, Vol. XXXII., p. 478.
[2] Borzyszkowski: *Inaug. Dissert.*, Greifswald, 1877.
[3] Wolff: *Inaug. Dissert.* Greifswald, 1899.
[4] M'Kendrick and Snodgrass: *Brit. Med. Journal*, 1891, 6th June, p. 1215.

regulating nerve centre in the brain becoming seriously affected by the deprivation of oxygen.

Attention has already been drawn to cases of after-damp poisoning in which the temperature rose very high before death. A like condition is not infrequently found in poisoning by illuminant-gas and charcoal fumes. Bloch [1] mentions four cases in which increased bodily temperature was found; in one case, indeed, it reached 39·5°C, and continued thus for four days. Klebs [2] observed the case of a man, twenty-three years of age, who was poisoned by CO. When found he was unconscious, there was marked dyspnœa, tremor and spasm of the muscles, very rapid pulse, very cold skin, and a very high temperature (unrecorded). He died the same day. At the post-mortem examination, the optic thalamus, and the pyramids and olivary bodies of the medulla were found intensely red and hyperæmic. On the day of the poisoning, moreover, high temperatures have also been noted in poisoned persons by a few observers, as Frerichs,[3] who records a temperature of 39° to 40°C, and by Gnauck,[4] one of 40°C.

In a large number of cases it has been found when the patient is recovering, that there is a rise in temperature which may be considerable. Marthen [5] reported five cases in which the body temperature was elevated. Benson [6] reported the case of a student who was found in a comatose condition, and who remained unconscious for thirty hours. During this time the pulse was 140 and the temperature 103°F. There was profound muscular prostration, with pains in the calves of the legs and in the feet. The pyrexia persisted for six days, when the temperature became subnormal (97°F.) A fortnight after recovery a copious crop of boils appeared. Runeberg [7] mentions a case of illuminant-gas poisoning in which irregular fever set in on the third day and continued till the seventh, although there were no complications to explain the rise. Jaksch,[8] in four cases of CO poisoning, took the temperature every two hours, while the patients were deeply comatose. The chart showed that the temperature was subject to many fluctuations, and that pyrexia was always seen, sometimes for only one to two hours, but in one instance up to 39°C. Jaksch also found subnormal temperatures, but he held the view that increase of temperature was one of the characteristics of CO poisoning.

In some of the cases in which a day or two after the accident fever

[1] Bloch: *Inaug. Dissert.* Leipzig, 1902.
[2] Klebs: *Virchow's Archiv*, Vol. XXXII. p. 454.
[3] Frerichs: *Vergiftung durch Kohlendunst.* Berlin, 1866, p. 107.
[4] Gnauck: *Charite-Annalen*, Bd. VIII, 1883.
[5] Marthen: *Virchow's Archiv.*, Bd. 136, p. 535.
[6] Benson: *Brit. Med. Journal*, 5th July 1873, p. 24.
[7] Runeberg: *Nach. Virch. Hirsch. Jahres.*, 1880.
[8] Jaksch: *Die Vergiftung*, 1910, p. 257.

has been found, this has been caused by the advent of sequelæ such as bronchitis, pleurisy, or pneumonia, one or other of which so frequently develops after CO poisoning; in other cases it may be due to reaction, the result of the vaso-motor disturbances; while in not a few cases, if we accept the toxæmic theory, it is the result of the action of toxins circulating in the blood. We have repeatedly insisted on the necessity of warmth in the treatment of patients suffering from carbon monoxide poisoning, and on the danger of exposing such patients to cold. This again we emphasise. It frequently has happened that conveying a patient into the cold air has made his condition much worse, and has in some cases been the determining cause of death.

The Urine in Poisoning by Carbon Monoxide.

Glycosuria.—Since Claude Bernard, Richardson, and others called attention to the presence of sugar in the urine of animals poisoned by CO, and since Hasse[1] found it in the urine of a man who was overcome by charcoal fumes, glycosuria has been described by a large number of German writers, some of whom go even the length of saying that it is a constant phenomenon. Friedberg,[2] for example, in his experiments on animals, invariably found it in those which were killed, and also in those which recovered. He also demonstrated its appearance in human beings who had been poisoned by CO. Senff,[3] who in 1865 published an important work on the relation of CO to the production of diabetes, found in his experimental work on dogs that sugar appeared very quickly after the inhalation of CO. In only one case out of eleven was no sugar found. In these cases poisoning occurred thirty to sixty minutes after the first inhalation of CO, and lasted two to two and a half hours, but never longer than three hours. He also found that if the circulation in the liver were checked, no sugar would appear. He regarded the appearance of sugar in the urine as due not to the altered circulation in the liver, but to the direct action of CO on the nervous system. Frerichs[4] found it in eleven cases out of sixteen, in one of which it lasted three days. According to Litten, glycosuria appears in about 70 per cent. of cases. Sir Thomas Oliver[5] is one of the few British authors who has noted its presence. He discovered glycosuria in a man who was poisoned by gas while turning up the soil in a roadway in which there had been a large fracture of the gas main. In such circum-

[1] Hasse: Quoted by Leudet. *Archives Générales de Méd.*, 1865. *Preuss. Med. Vereinszeitg. N. F.*, Bd. II. 1858, p. 176.

[2] Friedberg: *Die Vergiftung durch Kohlendunst*, 1866, p. 127.

[3] Senff: *Inaug. Dissert. Dorpat*, 1865.

[4] Frerichs: Schmidt's *Jahrbucher*, 1889, No. 9, p. 252.

[5] Thomas Oliver; *Diseases of Occupation*, p. 70.

stances the soil becomes so impregnated with gas for a considerable distance around the source of leakage, that not infrequently, while digging trenches, labourers are overcome. In this case glycosuria lasted three or four days. In both Oliver's and Frerich's cases the glycosuria lasted longer than usual. It generally disappears before the end of the second day.

As the urine in many cases of CO poisoning is only examined a day or more after the actual poisoning, sugar although present earlier may thus be missed. In a case recorded by Jaksch [1] it disappeared in eight hours. This may explain why so few authors relatively have noted its appearance. One of the few French writers who have observed glycosuria to follow CO poisoning is Ollivier,[2] who observed it in one case only, and regarded its appearance as unusual. Brouardel,[3] who had great experience of such poisoning cases, never once encountered glycosuria in man, although he frequently found it in experiments on animals. Kobert [4] holds that sugar only appears in the urine in the very acute cases, and that it is never produced in those cases in which there has been gradual poisoning. Out of fifteen cases, Bloch [5] found that of four which were fatal, in one only was sugar demonstrated in the urine. In five of the cases which recovered it was also present, but in two of these only in a very small amount. Kahler [6] found sugar only in serious cases; and Jaksch [7] and others arrived at the opinion that the amount of sugar was proportional to the severity of the poisoning. This view, however, has not been confirmed.

It is well known, since the experimental work of Claude Bernard, Richardson, and of those whose names we have already mentioned, that glycosuria can be produced experimentally in animals by asphyxiation and by exposure to CO. After the animals recover consciousness, they pass large quantities of pale urine, followed by the appearance of sugar, which very soon disappears. In animals it has been demonstrated that much more sugar is passed than in man.

But other conditions must be at work to account for the great difference in the results between experimental and clinical research, for Borzyszkowski,[8] even when he pushed the poisoning in his experiments on dogs to very grave limits, in some cases gradually poisoning them for

[1] Jaksch: *Prager Med. Wochenschr.*, 1882, Vol. VII., p. 161.

[2] Ollivier: *Archives Générales de Méd.*, 1879, p. 513: also *Bulletin de l'Academie de Méd.*, 1889, p. 173.

[3] Brouardel: *Les Asphyxies par les Gaz., etc.*, p. 30.

[4] Kobert: Schmidt's *Jahrbucher.*, Bd. CLXXXVIII., 1880.

[5] Bloch: *Inaug. Dissert.* Leipzig, 1902.

[6] Kahler: *Prager Med, Wochenschr*, 1881, Vol. VI., p. 474.

[7] Jaksch: *Zeitschr. f. Klin. Med.*, 1886, Vol. XI., p. 20.

[8] Borzyszkowski: *Inaug. Dissert. Greifswald*, 1877.

weeks, seldom found sugar. Again, other experimenters as Biefel and Poleck[1] found it occasionally when poisoning was very acute, or where this had been going on for some time; and Araki,[2] in his experiments on animals, showed that when the poisoning was induced in those which had been starved, no sugar will appear. *He found that the results on dogs, rabbits, and chickens after inhaling carbon monoxide gas are the same as those found after inhaling air which was very poor in oxygen.* In cases of well-fed animals poisoned by CO, sugar, lactic acid, and albumen appeared in the urine, due, he believed, to insufficient oxidation, from the blood being poorer in available oxygen.

An interesting point in connection with CO poisoning was brought out by Eckard,[3] who demonstrated that while in almost all forms of diabetes subcutaneous injections of chloral made the sugar diminish or disappear from the urine, this was not so in the glycosuria of CO. In poisoning by CO, we have in several cases made a very careful examination of the urine passed during the first few days, but we have never been able to demonstrate the presence of sugar.

The whole subject of the appearance or non-appearance of glycosuria in CO poisoning had thus become indefinite; but the recent most careful experiments of Munzer and Palma[4] have, it seems to us, placed this subject on a sounder basis. At the outset, these experimenters confirm the fact, insisted on by several observers since Araki, that oxygen-starvation leads to degeneration of the albuminous constituents of the body. They affirm that the key to the question of the presence or absence of sugar in the urine after CO poisoning is to be found in the circumstances under which the poisoning takes place. They confirm Araki's view that no sugar will appear in the case of starved animals. They demonstrated that the same thing occurs in man. They also found, in one case of CO poisoning in a man, a considerable amount of lactic acid on the first day, a little on the second, and a mere trace on the third. They hold that sugar may not be seen in mild cases of poisoning, as in these there is not the same extent of disintegration of albumen in the body. In such cases they found that the giving of grape sugar would not make any sugar appear in the urine. But in most cases of CO poisoning, they maintain, there is glycosuria, and that this can generally be produced by giving grape sugar (alimentary diabetes). In some cases glycosuria disappears very quickly, and in one case sugar was given for nineteen hours without producing it, and their view is that everything points to the conditions of

[1] Biefel and Poleck: *Zeitschr. f. Biolog.*, 1880, Vol. XVI., p. 359.

[2] Araki: *Zeitschr. f. Physiol. Chemie*, Vol. XV., 1891, p. 364, also Vol. XVI., 1892, p. 453; Vol. XVII., 1893, p. 311.

[3] Eckard: Quoted by Kobert. Schmidt's *Jahrbucher*, December 1880.

[4] Munzer and Palma: *Zeitschr. f. Heilkunde*, 1894, Vol. XV., p. 185.

the particular case as determining the production of alimentary glycosuria and the times of its disappearance.

Glycosuria may be caused by irritation of the so-called diabetic centre in the medulla, either directly produced on the centre itself or indirectly through the afferent nerves; or it may be due to some direct action of certain substances on the hepatic cells leading to a rapid transformation of glycogen into sugar. If we accept the toxæmic theory of CO poisoning, this would go a long way towards explaining the appearance of sugar in the urine. It is also known that temporary glycosuria follows asphyxia and other conditions.

Hæmaturia has occasionally been noted after poisoning by CO. In a case reported by Wolff,[1] the patient on the first day passed urine with a considerable amount of sugar, albumen being also present. But four days later he passed a large quantity of urine with much blood in it, the temperature that night being 40·1°C. There was no other attack of hæmaturia, and the patient made a good recovery.

Albuminuria.—Albumen may frequently be found in the urine for a few days after poisoning by CO. German writers declare that it is a frequent accompaniment of the glycosuria. Theilemann[2] found it present in twenty per cent. of the cases. We have seen it once after CO poisoning by producer-gas. Frankel[3] was the first to point out that after CO poisoning there is increase in the amount of urea excreted. He believes that owing to the want of oxygen the albuminous constituents of the body disintegrate in a marked manner, giving rise to the production of increased quantities of urea.

Polyuria.—Many writers have described the passing of large quantities of clear urine for a day or two after recovery from CO poisoning. For example, Motet,[4] in his report of his own symptoms of poisoning, mentions that for three days he passed very large quantities of urine in which there was neither albumen nor sugar.

Anuria has also been noted. Thomson[5] reports a case of CO poisoning caused by the patient being in a small room in a dredger with a smouldering fire, in whom there was *partial suppression of urine.* At the end of the first day he passed only 7 oz. of urine, on the second day 15 oz., on the third day 23 oz., and afterwards normal quantities, no sugar or albumen being present, and the urea being in normal amount.

[1] Wolff: *Inaug. Dissert*, Greifswald, 1899.
[2] Theilemann: *Inaug. Dissert*, Halle-Wittenberg, 1903.
[3] Fränkel: *Virchow's Archiv.*, Vol. LXVII., 1876, p. 273.
[4] Motet: *Annales d'Hygiène Publique*, Vol. XXXI., 1894, p. 258.
[5] Thomson: *Lancet*, May 6th, 1903.

Effect of Carbon Monoxide on Respiration.

It is now well recognised, owing to the action of CO on the blood causing decreased corpuscular supply of oxygen and thus leading to decreased tension of CO_2 in the blood, that the respiratory centre becomes crippled, and that, owing to its action on the nervous mechanism of the heart or on the circulatory centre in the brain, *i.e.* the root of the vagus, cardiac movements are profoundly affected. The fact, then, of CO having such an action on that part of the nervous system which is of primary importance to life, explains the serious nature of CO poisoning and why sudden death may frequently take place. In fact this action can be compared to that of chloral and morphia, which also affect most seriously these important centres of respiration and circulation in the medulla.

The preliminary quickening of respiration found in the first stage of CO poisoning is soon lost, the breathing returns to its normal rate, at which it remains for a short time, and is thereafter soon followed by gradual slowing, becoming less rapid and more superficial. As the comatose condition deepens and as poisoning becomes more profound, characteristic pauses in the respiratory rhythm may be made out, these gradually lengthening as the condition becomes worse. In many cases, then, breathing becomes slower and slower and more superficial, the movements of the chest become less marked and the pauses lengthen, till at last breathing entirely ceases. Death may come on quite quietly and slowly during or following one of these pauses. In other cases the respiration becomes very rapid and shallow, interrupted every now and then by long pauses or deep sighs, and grows weaker and weaker till it is almost imperceptible. In others, again, death may be preceded by four or five loud noisy inspirations. Occasionally it is found that the respiratory movements are very variable, being very quiet and slow for a time, then very rapid and energetic, to be followed by a period of quietness with the development of well-marked pauses, during which there is absolute immobility of the chest. In other fatal cases there may be a very peculiar type of breathing, a series of five or six quick, almost precipitate expirations, followed by a deep and noisy inspiration. The gradual decrease of frequency and depth of respiratory movements points to the gradual paralysis of the breathing centre, which becomes less and less susceptible to the normal exciting agent in the blood.

There has been considerable controversy over the question as to whether dyspnœa is a usual feature of CO poisoning. Undoubtedly the change in the character of the respiration will depend to a considerable extent on the fact whether poisoning has been rapid or slow. Where the percentage of CO is small, there may not be much alteration in the

rhythm and frequency of the breathing till poisoning is far advanced. Klebs[1] carried out a number of experiments on animals and found that where poisoning was gradual and the CO_2 formed was removed, the percentage of oxygen being kept up, there was only slight restlessness with 0·4 per cent. CO, but when this was increased to 0·8 per cent. that extreme restlessness set in till coma developed. The breathing up to this stage was not markedly affected, but then it became slower and slower, with distinct pauses till it stopped altogether, there being neither dyspnœa nor irregularity up to this point. The fact that there is no dyspnœa indicates the difference between poisoning by CO and asphyxiation; in the latter condition the organism seeks by increased respiratory movement to lead more oxygen to the lungs.

Very interesting work was carried out by Biefel and Poleck[2] which confirmed the work done by Klebs. They held, however, that charcoal fumes do cause dyspnœa because of the large amount of CO_2 present, but that in illuminant-gas poisoning there is generally found the typical action of pure CO, as the air does not become impoverished of its oxygen.

In several cases there is a form of *Cheyne-Stokes' breathing*. In one of our cases of producer-gas poisoning this was present. In this case, however, the man was addicted to alcohol. Dr Geo. H. Logan of Cleland, narrates the case of a man who had been drinking very heavily for a few days, and who, in a semi-drunken condition, started his work of cleaning out a conduit in the air of which a considerable percentage of CO was present. When he was poisoned, a very exaggerated type of Cheyne-Stokes' breathing came on at intervals, with slow and very irregular pulse, the rate of the pulse not being slowed or affected in any way during the periods of apnœa. The man frequently relapsed into an unconscious condition, when artificial respiration had to be resorted to. Such cases as these make, however, very rapid and perfect recoveries. Both alcohol and CO seem to act chiefly on the nervous system, and it would be difficult to say in these cases which of the poisons actually led to this condition; probably it was the result of their combined action. But alcoholics are particularly susceptible to gas-poisoning, and in many such cases we find marked and often exaggerated nervous symptoms.

There are very few cases of poisoning by CO in literature where this type of breathing has been recorded, although it cannot be rare. Theilemann[3] reported such a case. A strongly-built muscular man, with his wife and three children, was poisoned. The wife, who was least affected, woke up to find two of her children dead and her

[1] Klebs: *Virchow's Archiv.*, Bd. XXXII., p. 473.
[2] Biefel and Poleck: *Zeitschrift. f. Biologie*, Bd. XVI., 1880.
[3] Theilemann: *Inaug. Dessert.*, Halle, 1903.

husband unconscious. His breathing was irregular, and there was marked Cheyne-Stokes' respiration. Sometimes the respiratory rate was about twenty-five per minute, but more often very slow and intermittent. The Cheyne-Stokes' breathing gradually became less distinct, and on the fourth day it disappeared altogether. On the fifth day the patient developed bronchitis, pleurisy, and pneumonia of the bases of the lungs, and died two days later. Another interesting point in this case was that *CO was demonstrable by the spectroscope in the blood four days after poisoning.* We have already said that in some cases the breathing, instead of being slow, is rapid; but even here the characteristic pause is generally found, eight to twelve hurried, perhaps noisy, superficial respirations being followed by a prolonged pause. In some cases the pause is periodic, occurring at regular intervals, but in others there is no real cycle. In some cases with pronounced nervous symptoms Cheyne-Stokes' breathing may be looked for. Chauffard [1] reported a case of what he regarded as toxic encephalitis following CO poisoning in which there was coma vigil, rectal incontinence, and retention of urine which lasted three days, along with signs of meningeal irritation, notably Kernig's sign, paresis of the upper limbs, and Cheyne-Stokes' breathing.

In connection with this type of breathing, produced in certain cases by CO, and also with the development of Cheyne-Stokes' breathing, Mosso[2] has pointed out that these are often met with in persons who have ascended to great heights. Indeed, the effect on the respiratory movements of breathing rarefied air is most interesting when considered comparatively with CO poisoning. Mosso has made some very interesting observations with regard to respiration in rarefied air. Following the description of Saussure after his first ascent of Mount Blanc, most observers held that as the air became more rarefied, the lungs, in order to make up for this, acted more frequently and more vigorously. But Mosso found on the contrary that breathing in high altitudes neither increased in depth nor in frequency, and that both were even diminished. He found, for example, that the respiratory rate at a height of 4560 metres was 8 or 9 per minute. "At the height of 3337 metres a much smaller quantity of air is breathed than in Turin, etc. This fact is interesting, showing as it does that there is in man a respiration of luxury, as I have termed it; at lower levels and at sea level, we breathe a quantity of air which surpasses by far the needs of the body." Taking observations on himself regarding breathing at a height, Mosso found that of the respirations, the third is less than the second, the fourth and the fifth becoming still more superficial, while at a certain point, indeed,

[1] Chauffard: *Journ. des. Pract.*, 18th March, 1913.

[2] Mosso: *Life of Man in the High Alps*, p. 31, loc. cit, p. 46.

the thorax tends to become motionless. In others he noticed exactly the same thing, viz., that in breathing at a height the respiratory rate tends to diminish, and also that there are periodic pauses. These pauses increased with the altitude and as the amount of oxygen of the air decreased. "The breathing," he says, "sometimes continued for hours with this rhythm: three descending movements of which the first is forcible and the other two or three weak, being followed by a pause which lasted regularly twelve seconds before the return of another series of three descending respirations."

In certain cases of gas-poisoning this very same type of respiration has been noted, the analogy thus being very striking. Mosso holds that the Cheyne-Stokes' breathing was due to diminution of CO_2 tension in the blood, for it disappeared when CO_2 was administered by inhalation; indeed, he regarded this as the primary cause of mountain sickness. Pembrey and Allan[1] also made Cheyne-Stokes' breathing disappear by administering CO_2. In fact, it is now recognised by most physiologists that Cheyne-Stokes' breathing is brought about by diminished tension of the CO_2 in the alveolar air, and that in CO poisoning as in breathing rarefied air, owing to oxygen-starvation, there is a decrease in the CO_2 tension in the alveoli. "In high altitudes the CO_2 in the air is less, and this, together with the increased pulmonary ventilation produced by oxygen-hunger, lowers the alveolar CO_2 tension. The respiratory centre thus lacks its normal excitement. The CO_2, moreover, in the venous blood, under ordinary circumstances, raised the oxygenation by increasing the dissociation of OHB" (oxyhæmoglobin).[2]

Geppert[3] carried out a large number of experiments to discover the different effects on respiration of breathing CO and air gradually depleted of its oxygen, or, in other words, of slow suffocation. In both cases there is less oxygen inhaled, less oxygen in the blood, and therefore less oxygen consumed in the tissues. He found that breathing is altered in a very distinct manner as soon as air containing 7 to 8 per cent. of oxygen is reached; the animal breathes three or four times the normal quantity; the inspirations are increased and more pronounced; further, the consumption of oxygen sinks in the most profound manner. The breathing becomes increased sooner than the consumption of oxygen becomes diminished. Now if an animal is poisoned by a moderate quantity of CO, for example 0·5 per cent., the different stages are easily followed. Poisoning is much more rapid than in breathing oxygen-impoverished air. After a certain time the consumption of oxygen sinks

[1] Pembery & Allan: Quoted by Leonard Hill. *Recent Advances in Physiology.*

[2] Leonard Hill: *Recent Advances in Physiology.*

[3] Geppert: *Kohlenoxydvergiftung und Erstickung.* *Deutsche Medicinische Wochenschrift*, 12*th* May, 1892, p. 419.

as low as in asphyxia. The animal breathes much the same as ordinarily, sometimes more strongly, sometimes less strongly. But in CO poisoning of gradual causation, the respiratory centre does not respond by increase of breathing to the want of oxygen, the important point being that the respiration in CO poisoning is much shallower than in suffocation. Geppert insists that these facts can only be explained by holding that CO has a particular specific action on the nerve-centre.

Where coma has been at all deep, extensive moist râles are generally found in the chest. These are apt to become exaggerated when breathing is very quiet, disappearing or becoming much less when breathing becomes stronger. They are often the result of œdema of the lung caused by the circulatory changes. It is well to remember that CO appears to have a different effect on the breathing in different people, for when several persons have been exposed to the same gas under the same conditions and for the same period of time, it is found that in some the breathing is snoring or noisy in character, while in others it is very quiet.

Effect of Carbon-monoxide on the Cardio-Vascular System.

In many cases it is principally on the heart that the effects of the poison fall, thus accounting for the overwhelming depression, prostration, and loss of vitality complained of in so many cases of CO poisoning. Klebs believes that the greater resistance of certain persons to CO depends upon a greater capacity and strength of the heart in combating its injurious action on the vascular system; and it has already been demonstrated how much more susceptible to CO poisoning than healthy persons those are who suffer from certain cardiac lesions. In many cases of CO poisoning although no organic lesions can be made out, the heart sounds suggest weakness of the cardiac muscle owing to the action of the poison on its nervous mechanism.

The following is a case recorded by Lewin[1] in which very little could be made out, but it is of interest from the legal point of view. A woman was working in an ironing room where producer-gas was used for heating the irons. One day she vomited twice on the way home, and complained of pains in the head, stomach, and back, and of nausea and giddiness. The heart was very weak, the pulse being small, irregular, and frequent, and the hands and feet cold. Her temperament during this time became changed, and she could not leave her bed. At first no compensation under the Compensation Act was allowed. One of the medical experts who was called at the time held that there could only be gas-poisoning when the poisoning was acute; that if there were no acute

[1] Lewin: *Berliner Klinische Wochenschrift*, 28th October, 1907, p. 1367.

symptoms during the period of stay in the poisoned atmosphere then no further effects could be attributed to the poison. Such a view, however, is quite incorrect. Lewin says that the first contention only refers to the symptoms, not to the gas-poisoning, and it is moreover incorrect. The time of development of the symptoms depends upon the individual. In this case the patient was not ill when she left the laundry; but Lewin holds that this cannot be adduced as a reason that the symptoms which developed soon after she left the work were not due to gas poisoning.

In many such cases, he holds, serious effects are not found at the time The person affected may have been near an open window, and consequently the CO was not able to produce its worse effects at the time of working. But this observer misses two very important points in the production of the symptoms after the patient left the laundry, viz., the fact that very often the symptoms of CO poisoning develop or become worse when the patient is exposed to the cold air; and second, that muscular exercise, such as walking, makes them very much more apparent. The case came before the High Court, and Lewin was asked the following points. Is it really the case that the woman was unable for work for thirteen weeks? Yes. 2. Can you say that the woman was ill before, was hysterical, and that the accident made her worse?—No. 3. The reason?—CO poisoning. Lewin examined her six months afterwards, and found her still suffering from headache and oppression in the chest whenever she exerted herself. She was pale and weak. The heart was beating very quickly, the pulse rate being 125-130 per minute; in fact, tachycardia had been marked from the beginning. The Court held that she was suffering from the effects of CO poisoning, and she was awarded compensation. Recovery in all such cases, where no organic lesion is discoverable, is remarkably slow, and in certain cases permanent cardiac derangement results.

Medical practitioners in large Iron and Steel Works, who have had considerable experience of gas-poisoning cases since the time when gas first began to be used for various purposes in these industries, have informed us that they were often struck by the serious cardiac conditions produced by repeated gassing. Dr Fotheringham of Motherwell, for example, saw many cases fifteen or twenty years ago when the manufacture of the gas at the steel works was more defective than now, and when its poisonous nature was not sufficiently appreciated by the men. He came to be expert in diagnosing these cases by the peculiar lividity of the face, the breathlessness and oppression about the chest, "and the pulse all wrong." Young men were generally most affected; indeed, many strong young men fresh from Ireland were reduced to wrecks at the end of a few months.

Owing to the action of CO on the nutrition of the cardiac muscle, slight dilatation is often seen. Dr Haldane has seen cases of this after partial poisoning by CO, and also symptoms very similar to those of heart disease persisting for weeks or months. We have occasionally had cases in which the symptoms complained of were principally cardiac, but where neither the slightest dilatation nor any organic lesion of the heart could be detected, the only sign that anything was wrong being a slight muffling of the sounds, which were not so clear and distinct as normally. These patients complained of breathlessness on exertion, præcordial distress with feeling of oppression in the chest, palpitation with irregular and greatly increased heart-beat, the slightest exertion sending it up to 120-140 per minute. Marked pulsation of the large vessels was a very characteristic sign in the greater number of these cases. Fainting attacks were frequent; indeed, in serious cases of CO poisoning it has been found that these attacks may come on at intervals for weeks after the accident. Even in minor cases of poisoning in very susceptible persons, owing to the action of the heart, fainting fits may come on. There was a remarkable epidemic of fainting among girls in a school in the Midlands, where in six weeks forty-six cases occurred. There was no apparent cause at first for this, but it occurred only in one room in the school, and although no leakage from gas-pipes could be discovered, the outbreak stopped as soon as the floor was laid with asphalt. It was supposed that the cause of the poisoning was CO, probably escaping from an underground fire through the fissures in the strata caused by a recent earthquake.

The cardiac symptoms complained of after CO poisoning are tightness and oppression about the chest and præcordial region, pulsation in the carotids, and in some cases in other large arteries of a most uncomfortable character, palpitation of the heart which interferes with the patient's sleep, and attacks of tachycardia. There may be marked irregularity of the heart's action. These symptoms may be accompanied by a feeling of "goneness" (this is a miner's word for the feeling). Colonel Elsdale,[1] after being poisoned by coal-gas used for inflating balloons, complained for three days of constriction about the chest. He writes:—"My heart felt as though it were held in a vice or imprisoned in an iron band, and one could not draw a deep breath without pain and difficulty."

Sanger Brown[2] reports the case of a man of 34 who was seriously gassed, being rendered unconscious for three days, but who was able to go about in three weeks. For a time he suffered on the slightest

[1] Lt.-Colonel Henry Elsdale; "Resuscitation by Oxygen," *Nineteenth Century*, 18th May, 1891, p. 721.

[2] Sanger Brown; *Journal of the American Med. Assoc.* Chicago., April 28th, 1906, p. 1265.

exertion from dyspnœa. His expression was dull and stolid, and his face purple. His pulse rate was 140, and for a long time it varied from 110 to 140, but was nearly always over 120. There was no interference with motion or sensation. The events of the period of 36 to 48 hours previous to the accident had been erased from his mind, but his memory for the events of his early life remained good. He now forgot, however, almost immediately, the daily events in his present life. By dint of practice he remembered his way about the house and the name of the nurse in attendance. He was very emotional and constantly complaining. He could read, but could not discuss current topics. His condition remained stationary for ten months, until a fortnight before he died when he grew worse, suffering from severe attacks of dyspnœa. Death occurred suddenly. Tachycardia was a marked feature in this case. Sanger Brown explains it by assuming an interference with the nutrition of the neurons which naturally regulate the heart's action. Some of the cases of tachycardia may be the result of a neuritis of the pneumogastric nerve with trophic disturbances in the heart muscle and cardiac dilatation.

We have considered under sudden death the fact of heart failure being a not uncommon cause. An engineman in charge of a gas-engine plant was overcome by gas. After partially recovering, he was able to proceed home, where he expired suddenly three hours afterwards. The jury brought in a verdict that death was due to failure of the heart accelerated by water-gas poisoning. This, we may add, is the usual verdict returned in such cases.

The *condition of the pulse and blood pressure* during the different stages of CO poisoning may now be considered. After breathing an atmosphere of CO for a very short time, there is a considerable rise of blood pressure, probably due to contraction of the middle coat of the arteries. There is marked throbbing of the vessels, uncomfortable feeling of fulness in the head, oppression and constriction about the chest, with, perhaps, slight giddiness. The respiration at the same time may be increased. There is a considerable increase in the frequency of the pulse, it may, indeed, be doubled, the heart beats more irregularly, six or seven pulsations occurring in what one might term a precipitate manner and then succeeded by perceptible pauses and, later, by irregular and rapid beatings.

Between this and the next stage, although there is no real boundary line to divide them, it is sometimes found that the heart beats alternately rapidly and slowly. Then follows a decrease in frequency, which sometimes may be marked. We have seen it as low as 40 per minute, and various observers have noted it even less frequent, while at the same time the pulse character is soft and easily compressed, not unlike the pulse

observed in enteric fever. Pokrowsky[1] considers that this second phase in the phenomena of the circulation in poisoning by CO has all the characters of a blood pressure curve due to peripheral irritation of the vagus; and that the increased action in the first stage is due to the irritation of the cardiac centre in the upper part of the spinal cord. Traube[2], however, considers the increased frequency in the first stage to be due to the irritation of the vaso-motor centres, because it occurs in cutting the vagus notwithstanding destruction of the cerebro-spinal system. With this decreased action of the heart there are slower respiratory movements, restlessness, dilatation of the pupils, and a certain diminution in the body-heat.

During the third stage the blood pressure is low, the pulse again becomes increased in frequency, but is small, thready, often irregular, and almost imperceptible, and there is loss of the first cardiac sound. This stage, in which the involuntary muscular fibres of the whole organism and the fibres of the heart are paralysed, points to imminent danger, for unless the organism is quickly supplied with oxygen death will result. Pokrowsky found that in this stage irritation of the medulla oblongata, or of the cervical cord, was followed by considerable increase in blood pressure. This stage is probably due to paralysis of the vaso-motor centre. Just before death the heart may beat very slowly. It has sometimes been found that there may be only two or three beats in ten seconds, but following after a deep inspiration or sigh the heart beats very rapidly, and then stops altogether. In some cases, just at the moment of death, Artigalas[3] found that the heart seemed to make a last effort, so to speak, because following very slow beats there came a variable number of precipitate pulsations, then a pause, and death.

Attention has already been drawn to the curious fact that during the pauses in respiration which are frequently found in severe cases of poisoning by CO, the pulse rate is not affected at all, although one would naturally expect it to be slowed. In connection with the condition of the pulse during the period of apnœa produced by breathing very rarefied air, Mosso[4] writes: "It is a proof of the incipient paralysis of the nerve centres that during the respiratory pause the heart becomes insensible to the action of carbonic acid. If we observe the cardiac pulsation during the period of repose of the thorax, we see that the pulsations are all equal, whereas so long an arrest of the breathing ought somewhat to slacken the last beats." In serious cases of CO poisoning, it must never be forgotten that the heart generally beats for a perceptible period after

[1] Pokrowsky: *Archiv. für Anat. und Physiol.*, 1866, p. 154.

[2] Traube: *Gesammelte Beiträge zur Path. und Physiol.*, 1871, Bd. I., p. 68.

[3] Artigalas: "Des Asphyxies Toxiques," *Thèse de Paris*, 1883, p. 69.

[4] Mosso: *Life of Man in the High Alps*, p. 45.

respiration ceases, and that as long as the heart beats, attempts at resuscitation must be carried out. These may prove successful, nay, have been successful in many cases. When the patient is recovering, the pulse becomes stronger and fuller and beats much more frequently. Marthen[1] found that where reaction sets in, the pulse is not unlike that found in enteric fever.

Susceptibility to CO Poisoning—Tolerance.

We have come across men both in ironworks and in collieries, who were able to work in an atmosphere which others could not face, owing to the bad air producing distressing symptoms in a very short time. In connection with ironworks, where gas is largely used, this fact is well known; some men who are recognised as proof against gassing being sent to those jobs where gas is present in considerable quantity. Not that it is thought that any man is absolutely immune to poisoning by gas, but experience has shown that these men are able to work in an atmosphere which would immediately knock over more susceptible men. We also know of men who have been gassed time after time, men who would go anywhere and who were seemingly proof against injurious atmospheres of CO in which other men could not work, and yet who recovered in a surprisingly short time, never indeed being longer off work than for a few hours, nor apparently suffering from after-effects.

Certainly in most cases the length of exposure, the percentage of CO present, and other conditions such as presence of CO_2 and diminished amount of oxygen, have an influence on the severity of the poisoning, but many cases are on record where very short exposure to small quantities of CO has caused death, so that idiosyncracy or individual susceptibility seems to play a not unimportant rôle. It has frequently happened that of a number of persons who have been exposed to the same poisonous atmosphere for the same time and under the same conditions, some have escaped while others have died; and in experiments carried out on animals the same has been observed by many authors. Again, a person may have been very seriously poisoned and yet make a very rapid and complete recovery, while another who has not shown at the time serious symptoms of poisoning, but has not lost consciousness, may develop very serious sequelæ and may even die.

It is an open question whether women can withstand CO poisoning better than men. Certain observers, with regard to charcoal and illuminant-gas poisoning, hold firmly the view that they can and do so, for they have found that women recovered after longer exposure

[1] Marthen: *Beiträge zur Kenntnis der Kohlenoxydvergiftung. Virchow's Archiv. für Path. Anat.*, Bd. 136, p. 535.

than men who had succumbed. In cases of poisoning by leakage from a coal-gas pipe in a dwelling-house, it is not infrequently found that the woman has been in the house for a few days, and that a small leakage has been going on continuously, but that when this had accumulated in sufficient quantity to do its deadly work, the men who were overcome died, while the woman, who had been longer exposed, recovered. In these cases, of course, the question of tolerance to the gas having been to a certain extent established, must not be lost sight of. The following case narrated by Tourdes[1] may be cited. A family in Strasbourg, which consisted of six persons—father, mother, two sons, a daughter, and a servant—breathed for forty hours an atmosphere impregnated with coal-gas which had escaped from a leak in the main in the street and had found its way into the different rooms of the house. Four were found dead, and the father and mother still living but unconscious. The former died, but the latter recovered, notwithstanding that she had been exposed for a longer period than any of the others to the poisonous atmosphere. For three days before the fatal accident, the mother had complained of symptoms which gradually became more severe, viz. :—headache, nausea, vertigo, and great weakness. This exposure during these days had probably led to a certain amount of tolerance being established. The same is true in the Zola case; Madame Zola recovered although her husband died. In a recent instance seen by one of us (Glaister), of a man, his wife, and a canary, who occupied part of the ground floor of a tenement, into which in the night illuminating-gas had entered by way of the soil and the flooring from a burst gas-pipe outside of the dwelling, the man and the bird were found dead, but the wife, though unconscious, recovered. The spectrum of CO was found in the blood after the *post-mortem* examination which was held on the body of the man about thirty-six hours after death.

A case by Theilemann[2] shows how other conditions come into operation, and go a long way to explain the apparent or supposed susceptibility of some individuals to CO poisoning. A man, his wife, and three children were poisoned by CO in a new house. Two of the children were found dead, one eight months, the other four years old, and while the father died on the seventh day, the mother and the remaining child of six years were hardly affected. There were two beds in the room both equally distant from the window. The man and child of four years occupied the bed nearer the fireplace; while on the other bed, which was against the gable-end of the house, lay the mother, baby, and six-year-old boy. It was found that there was neither plaster, paper, nor

[1] Tourdes: *Relation Médicale des Asphyxies occasionées à Strasbourg par le Gaz de l'éclairage.* Paris, 1847.

[2] Theilemann: *Inaug. Dissert.* Halle-Wittenburg, 1903.

oil paint on the wall against which this second bed was placed, so that the ventilation was probably much better.

The differences of the effect of CO in many cases could be explained in a similar manner. When Zola[1] died, poisoned by charcoal fumes from a stove, his wife escaped, although she breathed the air longer than her husband. But Zola had risen from his bed during the night and was found lying dead in the middle of the room. Probably in this case the slight exertion of getting up had been the cause of death. In many other cases the man has recovered while the woman has died. Castleman[2] reports a case where husband and wife were poisoned by charcoal fumes. The husband woke up early, arose suffering from slight headache, and left the room without noticing that his wife was lying dead in bed. Poelchen[3] reports another in which man and wife were exposed to CO under the same conditions. It was found that there was very little wrong with the husband, while the woman was unconscious for two days and died two months later. Glaister[4] also mentions a case where a family, who occupied a cellar-dwelling, were poisoned by coal-gas which had found its way into the house through the foundations of the building and the flooring, from a burst main. When discovered the mother and children were found to be dead, but the father alive although deeply comatose. A paraffin oil lamp in the room was found to be burning freely. In this case, however, the in-burst of gas had been equally sudden for all. Many cases could, however, be quoted to support either view, since much undoubtedly depends upon the constitution of the individual, whether male or female.

With regard to chronic poisoning, our own view is that women stand chronic poisoning by small doses of CO very much worse than men, that nervous sequelæ, for example, are very much more liable to develop in women. Toleration seems to be established very much sooner in men; and in factories and workshops into which gas (it may be from the producer of a suction-gas engine) has found its way, it is generally the women who complain first and who suffer most severely.

Although cases are on record in which sick persons have been exposed to CO along with healthy individuals and have escaped with their lives although the latter were seriously affected, there can be little doubt that any disease, more especially where there is a lesion of the heart or of the lungs, renders the individual more susceptible to CO poisoning. Claude Bernard[5] from his experiments found that sick

[1] Death of Zola: *Brit. Med. Journ.*, 4th Oct., 1902.

[2] Castleman: *Gazette des Hôpitaux*, 1892.

[3] Poelchen: *Berlin Klinische Wochens*, 26th June, 1882, p. 398.

[4] Glaister: *Medical Jurisprudence and Toxicology*, 1910, p. 652.

[5] C. Bernard: *Leçons sur les Effects des Substances Toxiques*, 1857, p. 197.

dogs resisted CO better than healthy ones, and Laroche [1] has reported an instance where a strong, healthy young girl of twenty years was exposed to CO, along with a girl of the same age who had been suffering for some time from typhoid fever and who had just had a severe attack of menorrhagia, in which the latter was able to shout for assistance and was little affected, while the healthy girl developed hemiplegia after being unconscious for a considerable time. Plenio [2] also relates a case in which an alcoholic woman, aged 56 years, died, and a girl who was suffering from chlorosis recovered. Eulenberg also believes that weakly people recovering from illness stand CO poisoning better. Moreover, chronic alcoholics stand CO poisoning very badly.

Persons with any heart lesion, more especially if there be any degenerative change in the cardiac muscle, are not only much more susceptible to CO poisoning, but they stand exposure to that gas very much worse. The after-effects in these cases may be very serious. Exposure to CO has been found to light up any tendency to heart disease, as in the case of Sir C. Le Neve Foster, and lead to permanent crippling of that organ. We are of opinion that no one with any serious lesion of the heart, more especially with any degenerative changes in the cardiac muscle, should be allowed to work in bad air in mines, either in black-damp, where there is deficiency of oxygen and increase of CO_2, where frequent blasting operations are going on in a badly ventilated place, or where there is any possibility of gas-poisoning, as, for example, in cleaning flues in connection with blast-furnaces, steel works, and ammonia plants, and more especially in gas-engine houses where there is always more or less CO in the atmosphere.

It stands to reason that persons with any disease of the lungs will stand gas-poisoning very badly. They have only a portion of their lungs available to procure oxygen to oxygenate the tissues, and consequently it is difficult for them, even apart from noxious atmospheres, to keep their tissues in proper repair and for metabolism to go on in a perfect manner, the result naturally being that they become more susceptible to CO, since they have less hæmoglobin available for the important duty of carrying oxygen to the tissues.

Toleration of CO is very quickly established in connection with those who work about gas-engine plants where there is always a considerable amount of this gas present in the atmosphere. We had one patient, however, suffering from phthisis with considerable consolidation of the right lung, who, although he had been on duty in such circumstances for three or four weeks, instead of becoming acclimatised, as he had been told he would, found himself getting so ill that he was forced

[1] Laroche: *Thèse de Paris*, 1865.
[2] Plenio: *Archiv. für, Med.*, 1888, p. 299.

to give up the work. The symptoms of which he complained during that time were essentially those of CO poisoning. He quickly regained what he had lost when he started work in the open air. Ball[1] describes the case of a father, mother, and daughter, the last being sixteen years of age, who were suffocated in their room owing to a defective chimney. Both father and mother recovered rapidly, but the daughter, who suffered from phthisis, a part of her right lung being consolidated, suffered very severely, having had convulsions for a day and being unconscious for two days.

W. C. C. Pakes,[2] in his evidence before the Transvaal Phthisis Commission, held the view strongly that if miners suffered from silicosis they were undoubtedly much more susceptible to, and suffered much more severely from the action of bad air, fumes from blasting, etc., than healthy miners. We have already described a case by Cullingworth in which serious legal consequences followed, and where father, mother, and daughter were exposed to CO poisoning. The father and mother died, and the daughter recovered. The circumstances of the poisoning were so suspicious that the daughter was arrested on suspicion of causing the death of her father and mother; but it was proved that death was due to CO poisoning. In both the father and mother extensive lung lesions were demonstrated at the post-mortem examination, which probably explained why they had fallen easier victims to gas poisoning.

Those attacked by serious sequelæ may be predisposed by some weakness of the nervous system. This will explain some of those cases in which neuritis, for example, developed after a very short exposure to CO. It is well known that some persons are more susceptible than others to certain poisons, such as lead and alcohol.

Susceptibility to CO poisoning varies greatly in different individuals. In the great Courrières disaster where 1100 lost their lives, thirteen men were ultimately saved by the re-establishment of the action of the fan and the reversing of the air-current, which purified the air in their part of the mine. Cases are on record where a man on going down into black-damp in wells or shafts of disused pits, or into parts of old disused workings, has been overcome, and where another man going down to rescue him has also been overcome; when both were pulled out, the second man has been found to be dead, while the first, who had been longer in the poisonous atmosphere, recovered.

Besides, we may read of cases where a man has been working for a considerable time in a tainted atmosphere, either deficient in oxygen or impregnated with CO, and a fellow-worker coming perhaps to relieve

[1] Ball: *Brit. Med. Journal*, 1878, Vol. I., p. 562.
[2] W. C. C. Pakes: Report of Transvaal Miner's Phthisis Commission.

him, has been immediately overcome, or, again, where the first man after working for a considerable time may succumb, and his mate going to his assistance has been also overcome so quickly that he was unable to effect a rescue.

We have also found that strong young men are much more susceptible to poisoning than older men, and some foremen in works where minor cases of gassing are often met with, knowing this, will not allow young men to undertake work where they may be exposed to gas. Where a group of men has been gassed, as we have seen, it is generally found that the young men are most quickly overcome and suffer most. In the Snaefell disaster this fact was especially recognised and emphasised. Of course in these cases the deeper inspirations and the greater elasticity of the chest in the young must be taken into consideration, thus allowing them more rapidly to absorb a larger quantity of gas.

Another point not to be lost sight of in coming to any conclusion regarding susceptibility to CO is, that increased muscular exertion will lead to those so exerting themselves being more rapidly overcome. Again, a matter apt to be overlooked regarding the idiosyncrasy of persons in relation to CO poisoning, and which has a very important practical bearing on rescue work, is that anxiety, worry, or marked mental effort acts just in the same way as, only more quickly than, increased and excessive muscular exertion, and that the leaders in rescue work are often those who suffer most. Miners realise this perfectly when they are doing any operations in bad air: as, for example, taking out rails in a very badly-ventilated section which has to be abandoned. Whenever a man in such a situation begins to be "put about," he is much more affected than his more easy-going companions. The same effect of concentration of the mind causing early fatigue is seen in a cycle race, where the man who is pacing is much more quickly tired out than the man who is being paced, since the latter does not require to fix his attention so much, and hence his actions become more automatic.

Regarding age and susceptibility to CO poisoning, there is no doubt that the old and the very young withstand poisoning very badly. In the very old we often find faulty metabolism, deficient nourishment of the tissues, and certain changes in the blood-vessel walls, which would predispose them in a marked degree to those effects on the brain which must be regarded as characteristic of CO poisoning. In reviewing cases recorded by Simon, Poelchen, Cramer, and several others, in which gross lesions were found in the brain, a large percentage of these were found among elderly people. In connection with the very young there can be little doubt, notwithstanding cases which have been published to prove the contrary, that young people withstand gas poisoning very

badly. Eulenberg [1] mentions a few cases in which children apparently showed greater resistance than adults; and MacCormick [2] quotes a case where two adults who were exposed to CO died, while a child of five months was saved. These cases are quite exceptional, however, and most observers believe with Lancereaux [3] that it is almost always the case in those who are poisoned by CO, that those who have most need of oxygen, that is to say young people, succumb most quickly or are most seriously affected.

In opposition to this, it is interesting to remember Paul Bert's conclusions from his experiments on animals, viz.—that the young can withstand gas poisoning much better than the old. He thought that this was because the tissues of the young consume less oxygen than those of older animals. The following case (Apl. 1911) may be taken as typical of the results upon adults and young who are exposed together to CO. The janitor of a school in Ayr, on opening the boiler-house in the morning, found a man and a woman lying unconscious and the fourteen months' old child of the woman lying dead in her arms. The boiler-house, which was below the ground level, had been forced open during the night, the door of the furnace, the fire of which had been banked with coke and coal the night before, having been opened, and as there was practically no ventilation, the fumes were able to do their deadly work. The woman was charged under the Children's Act, pleaded guilty, and was sentenced to six months' imprisonment. Regarding the increased susceptibility of children to CO, Boehm [4] is very emphatic; "It is" he says "quite certain that children succumb very rapidly to the action of this poison."

Lancereaux, Faure, and others have pointed out that very small leakages of coal-gas may have serious consequences on very young children, and also give rise to conditions in them which are most difficult to diagnose. The first-named mentions the case of a strong, vigorous infant, nineteen days old, who fell into ill-health, took no food, constantly slept with its eyes half closed, with feeble pulse, and almost lifeless condition, all of which symptoms were owing to inhalation of minute quantities of CO.

Another point regarding susceptibility to CO is, that men who are alcohol drinkers stand gassing very badly. This is also the case when they are exposed to any bad air, as in cleaning out conduits, sewers, etc., cleaning out the exhausters, tubes, and flues in connection with furnaces, ammonia, or producer plants, and in pits. The men themselves are quick

[1] Eulenberg: *Loc. cit.*, p. 136.
[2] MacCormick: *Medical News*, 1891, p. 517.
[3] Lancereaux: *Bulletin de l'Academie de Méd.*, 1889, Vol. 21-22, p. 170.
[4] Boehm: *Ziemssen's Cyclopedia of Med.*, Vol. XVII., p. 464.

to notice this, and they are apt to say that "blood soaked with whisky sucks in or draws in the gas." Formerly in Scotland, and the practice may still be followed out in certain parts, it was the general custom to give men who were working in any bad air, or doing any dirty job, a glass of whisky each every two or three hours. These the men, needless to say, took (it was indeed part of the day's wage), although many of them knew perfectly well that by doing so they were weakening their resistance to the gas. Griedenberg[1] mentions a case where a woman and her son, a strong young man who was in a state of alcoholic intoxication at the time, were poisoned by CO, the mother recovering while the son died; and a number of similar cases might be quoted from records. We shall also see when discussing nerve lesions and mental and nervous disturbances in both acute and chronic poisoning by CO, that alcohol plays a most important rôle in rendering the patient more susceptible to such sequelæ.

Another point, proved beyond doubt from our own experience both in mines and in connection with ironworks, etc., and more especially now in connection with gas-engine plants, is that *tolerance* to minute doses of this gas may be established, men through time being able to work in atmospheres in which at first they could not have breathed without producing well-defined symptoms, which in time altogether disappeared. In connection with gas engines this is frequently seen. The men at first complain, more especially when they come out into the open air, of splitting headache, a swimming sensation, slight staggering after they have walked a distance, and a feeling of nausea. These symptoms, however, gradually disappear. In time they are able to work in an atmosphere which would in ten minutes or less render a person not so immune quite ill. These men may not show signs of chronic poisoning. In others, again, we find that this toleration never becomes established; they continue to work, as they are told by experienced men that the symptoms of which they are complaining will soon wear off, but in the end they have to yield and throw up their employment in the gas-engine house. Others again, although they continue to work continuously for days in bad air, soon become anæmic and suffer from palpitation, breathlessness on exertion, headache, and other symptoms of poisoning.

This establishment of tolerance by constant exposure to gas has been recognised for a long time, for we find Faure[2] in 1856 writing thus:—"It appears to me, and these preceding cases confirm this thought, that certain individuals by exposure again and again to the vapours of carbon become at length able almost with relative impunity to undergo exposure to them. I have often seen animals, which I have repeatedly

[1] Griedenberg: *Annales Médico-Psychologiques.* Paris, 1900, p. 67.
[2] Faure: *Archives Générales*, 1856, Vol. VII., 5th Series, p. 33.

poisoned, and poisoned very severely in order to study the state of the heart, etc., acquire such invulnerability that it was difficult to asphyxiate them." Sir Thomas Oliver, in his experiments, by gradually habituating dogs to CO, found that he was able to expose them to as large a quantity as 0·8 per cent. for several hours without bad effects. Mosso also was able to expose animals in whom toleration has been established to very large percentages of CO gas.

A very interesting example of this development of toleration was found a number of years ago in connection with an underground fire in a pit in Wishaw. Three miners continued to work in the vicinity of the fire which they had helped to build-off, but from which the poisonous fumes continually escaped. They were occasionally overcome, and at first suffered considerably, but as they were well paid they continued at work until they became almost immune. From time to time men, attracted by the good wages, started in their section, but they were never able to continue long, as they suffered so much from headache, giddiness, and vomiting, sometimes even being overcome altogether and rendered unconscious. Owing to certain circumstances these three men left this pit, and started work in another where the air was very good. They declared that at first they could hardly work and that they suffered considerably; to them it appeared, from their difficulty in doing their work, as if they had come from good to bad air and not from bad to good. They worked in this place for two months, when they returned to their former work, to find to their surprise, however, that they had lost their previous immunity and that they were now quite unable to stand the poisonous atmosphere.

Nasmith and Grahame [1] have carried out a large number of most interesting experiments on guinea-pigs to discover what ultimate action CO had upon the body when inhaled over lengthy periods, and in the course of these experiments they came to definite conclusions regarding this question of toleration to CO. A number of guinea-pigs were kept in a gas chamber into which CO was admitted in sufficient strength to saturate 25 per cent. of the hæmoglobin of the blood, some of them being kept there for months. The guinea-pigs had, therefore, during their stay in the chamber, only the use of 75 per cent. of their hæmoglobin for carrying oxygen to the tissues. Under these circumstances it was supposed that loss of weight, anæmia, and ill-health would ensue in time, but the experimentalists were surprised to find that this was not the case, that the guinea-pigs were just as lively and active as before, and that they actually put on weight. The blood at first showed degeneration of the red blood corpuscles, but on the third day normoblasts appeared, and thereafter a steady increase in the number of red blood

[1] G. G. Nasmith and D. A. L. Grahame. *Jour. of Physiology*, 1906, Vol. 35, p. 32.

corpuscles occurred till a maximum was reached in three or four weeks; that is to say, compensation had taken place by the blood-forming organs (as they do in high altitudes, residence in which has been found very beneficial in cases of anæmia) working with greater activity, and producing red blood corpuscles in much greater number till this compensation had been achieved. Indeed the action of CO on the blood-forming glands is practically analogous to what goes on after considerable bleedings where the blood-forming organs work with greater activity.

These investigators then carried out experiments by which the saturation of the hæmoglobin was gradually increased till, after two weeks at 35 per cent. saturation, it was raised to 45 per cent. Under this percentage the animals at first showed symptoms of such a large amount of oxygen-carrying hæmoglobin being thrown out of action by the CO, that they looked ill and lost weight. In four or five days, however, they again gradually recovered, when examination of their blood showed a great amount of degeneration of red blood corpuscles; but larger numbers of normoblasts again appeared, and in a few weeks there was an enormous increase in the number of red blood corpuscles, the blood becoming very thick. One or two normal guinea-pigs were now placed with some of these guinea-pigs in which tolerance to 45 per cent. had been established. Even with the temperature kept warm they generally succumbed in three or four days. But another guinea-pig, after three or four days' illness, from which it appeared to be dying, was removed to the fresh air, where at first it appeared almost dead, but it gradually came round. After eight hours it was put back into the gas box. During the next few days it appeared ill. On examination of its blood between four and seven days after it had been put back into the gas chamber, the red blood corpuscles were found so degenerated, many of the corpuscles being mere husks, that it was surprising that the animal could live. Normoblasts, however, appeared in great numbers. These experiments are of great interest from the practical points they raise, and indicate that no case, however apparently hopeless, should be given up.

Another important point brought out in these experiments was that CO poisoning is followed by a leucocytosis of the eosinophil and pseudo-eosinophil forms of blood cells which varied in intensity with the strength of the CO. Where the saturation is small, a moderate toxæmia involving an eosinophilia is produced, as is found in nearly all moderate toxæmias and post-febrile periods, but where the saturation has been high, the eosinophils disappear, as is observed at the height of most acute infectious disorders. These observers also found that where there was prolonged high-saturation, myelocytes and erythroblasts appeared, pointing to cells in the bone-marrow working with increased functional activity. The experiments show further how profound are the changes in the

blood, and lead us to expect similar serious changes in the cells of the central nervous system. This work, then, has a most important bearing on the ætiology of CO poisoning.

Susceptibility to CO poisoning, then, will depend upon the age of the individual, those at the extremes of life being most susceptible; upon the constitution of the individual, some being more susceptible to nervous sequelæ owing to some inherent weakness of the nervous system, it being important to remember that it is upon the nervous system that the brunt of the attack falls; upon the depth of the chest movements, young adults, owing to increased elasticity of the chest walls, inhaling more of the poisonous gas than middle-aged individuals; upon the strength of the circulation, and also, perhaps, upon the amount and quality of hæmoglobin: all of which must have a great deal to do with the absorption of oxygen.

Convulsions.

We have already considered convulsions developing in those exposed to after-damp, CO from pit-fires etc., in mines, and it has been noted that they are not frequently observed. Klebs[1] and Brouardel,[2] both of whom had a very large experience of CO poisoning, affirm that convulsions are comparatively rare, and that the evidence brought forward in support of their appearance is mainly of experimental origin. For example, such animals as dogs, on being exposed in a room containing a high percentage of CO, quickly developed convulsions. Brouardel agrees that convulsions may similarly take place in human beings where an individual enters suddenly a room impregnated with a large amount of CO; but he holds that in ordinary circumstances convulsions are not seen. He has never seen them in patients who have committed suicide by inhaling gas after lying down in bed, neither the position of their bodies nor the condition of the bedclothes pointing to their having had convulsions. Brouardel mentions, only to brush aside as doubtful, several cases in which convulsions were said to have developed. He quotes, however, a case by Seidel of a student who, wishing to find out the properties of CO, placed himself underneath a balloon containing this gas and began to inhale it. After a few inspirations he was seized with convulsions and was with difficulty brought round. Witter of Dublin, being desirous of finding out the effects of CO on the human organism when the gas was quickly and freely inhaled, took two or three deep inspirations of it. He at once became unconscious and lay as if dead for half an hour. On regaining consciousness he was seized with convul-

[1] Klebs: *Virchow's Archiv.*, Bd. XXXII., 1865, p. 470.
[2] Brouardel: *Asphyxies par les Gaz.* Paris, 1896, p. 26.

sions. Many such cases may be met with in the literature of CO poisoning.

Convulsions have been produced experimentally in animals by partial suffocation, by exposure to large percentages of CO_2 or CO, or by placing them in an atmosphere very deficient in oxygen. A number of observers who have carried out experiments on animals with CO, have found that spasms of the muscles, quivering of the skin, twitchings about the mouth, and trismus occurred, and also, in a number of cases in which convulsions followed, that they were very severe. Some regard convulsions as a regular occurrence in CO poisoning. They hold that in many cases where they were not found, the stage when convulsions might supervene had been passed before the patient was found; while in other cases that the reason why convulsions did not develop was that the poisoning had not been sufficiently severe. We must add, however, that many very severe cases of CO poisoning undoubtedly occur without the supervention of fits; and that in acute poisoning by large quantities of CO the brain centres are usually paralysed almost from the beginning, so that no convulsions develop. In a considerable number of cases of poisoning to a medium degree, however, there is a stage of cerebral excitement in which convulsions may be seen. Many cases are also on record where convulsions appeared during the stage of reaction, after the patient had apparently recovered from the immediate and direct effects of the poisoning. In reviewing the literature of the subject, we have found many cases of poisoning by producer and illuminant-gas and charcoal fumes in which convulsions were a marked feature.

From all the evidence, then, we can safely conclude that where the poisoning has been rapid, a large percentage of CO being present, convulsions may be met with. For example, a man employed in doing repairs about a gas main, who was suddenly exposed to large volumes of gas containing a high percentage of CO, suddenly fell down unconscious and developed convulsions. Barclay[1] reports the following rather curious case. A gas stoker, twenty-eight years of age, had been gassed, and was admitted to hospital thirteen days after his accident. He had previously always enjoyed the best of health. After being gassed he had twelve convulsive seizures in quick succession. In these, however, there was not total loss of consciousness, but there were spasms affecting the arms and legs, and also the facial muscles, causing clenching of the teeth. Six days after the accident, he suddenly lost consciousness and fell down in a fit, thus hurting himself. The day after admission to hospital the convulsions again returned. After admission he was affected every four or five minutes with loss of

[1] Barclay: *Lancet*, 1866, Vol. II., p. 523.

memory and speech and with giddiness, then the head was drawn back with a slight spasmodic action, and his arms became extended and quite rigid, while occasionally he was seized with spasms of the muscles of the throat. These fits passed off and he made a perfect recovery.

In another case of coal-gas poisoning reported by Bolton,[1] the patient was found unconscious, apparently dead, the face, lips, and ears were blue, he was foaming at the mouth, and there was also trismus. Twelve hours afterwards he presented all the symptoms of a man suffering from hydrophobia. Violent spasms occurred about every five minutes and were preceded by most painful signs of fear. He foamed from the closed mouth, the trismus being continuous throughout, even during the intervals between the spasms. He thumped his sides and body violently with his hands. The sight of water or even the sound of it was the cause of instant spasms. Blowing over him had the same effect. He got worse, and it took three men to hold him in bed. Opisthotonos now set in and he had seminal emissions. He could take nothing by the mouth. He had been bitten by a dog five days previously, but the dog showed no signs of rabies. He made a good recovery. Bolton, who had had considerable experience of rabies, thought that the symptoms in this case simulated rabies very closely.

Cases of convulsions appearing after CO poisoning, which, however, were probably hysterical in origin, are occasionally reported. Itzigsohn[2] describes the case of a girl of thirteen, who, with a few other pupils in a school, was poisoned by CO, and who developed epileptiform convulsions. At exactly the same time almost to a minute every eight days for the next three weeks she had another fit; then at intervals of seven days. These convulsions completely disappeared. He regarded the case as a pure neurosis. Becker, who refers to this case, thinks that it was due to hysteria in a girl who was at puberty, and who was markedly predisposed to nervous affections.

Rigidity, Muscular Spasm.

Patients are sometimes found, even in cases where there have been no convulsions, with considerable rigidity of the muscles of the body or of the limbs. A leg or an arm may be found rigidly flexed or stiffened in extension, or the muscles of the back or neck may be affected, even marked opisthotonos having been observed. To a less degree these spasms may attack only certain muscles or groups of muscles, in some cases the flexors, in others the extensors of the limbs. The mouth may be rigidly closed. In other cases, owing to the spasm of the pectoral

[1] Bolton: "Coal-gas Poisoning followed by symptoms simulating Rabies," *Lancet*, 19th March, 1898.

[2] Itzigsohn: *Virchow's Archiv.*, XIV., 1858, p. 190.

muscles, the arms are crossed in a forcible manner in the attitude of embrace, and sometimes the legs may be crossed and quite rigid.

In two cases reported by Millar[1] this feature was well marked. In the first the person lay unconscious, the breathing became stertorous, the pupils were contracted, the surface of the body cold, pale, and clammy, the conjunctivæ and mucous membrane bright red in colour, the pulse small and frequent, and the *legs rigidly crossed.* In the other case the *right arm was so firmly flexed* on the chest that it was almost impossible to bend it, the legs being also rigidly flexed. The pulse was 40 per minute, soft and compressible, and the breathing slow and laboured. In rare cases there has been noticed spasm of the psoas and other muscles, causing the patient to sit up rigidly. In these cases the lower limbs were rigidly extended. In other cases the patient may have clonic spasm of the muscles of the arms and legs, these spasms being transient in character and lasting only a few seconds. The muscles of the face may also be affected. Elgood[2] describes peculiar movements in a young woman. On regaining consciousness she exhibited persistent and regular semi-flexion and pronation of both arms, which produced movements as if she were rubbing her hands. Elgood regarded it as perfectly co-ordinated movement and one apparently accompanying her return to consciousness, as a quarter of an hour later she became more conscious. A periodic opening and closing of the eyes has also been noticed.

Marthen[3] reported the following case in which there was rigidity as well as clonic spasms. A man, aged 33, was poisoned by CO and became unconscious. On being lifted, the right arm was found to be rigid. The fingers of both hands were slightly flexed. The arms were approximated to each other but not crossed. The muscles of the right arm showed fibrillary contractions. From time to time there was clonic spasm of the muscles of rotation of the forearm. There was rigidity for a few seconds of both arms. The legs were bent at the hip and the knee-muscles were quite lax. By next day the muscular rigidity and spasms had disappeared. On the right hand there were a few cherry-red spots. The skin of the body showed slight icterus.

Muscular Tremor and Choreic Movements.

Very often, while the patient is passing out of the unconscious condition, there are irregular and, at times, spasmodic movements of the arms and legs, and for a few days after recovery tremor is often found. For example, when the arms and fingers are extended, marked tremor of

[1] James Millar: "Two cases of Poisoning by Carbonic Oxide," *Lancet*, 28th March, 1885.

[2] Elgood: "Poisoning by Carbon Monoxide." *Lancet*, 15th September, 1900.

[3] Marthen: *Loc. cit.* (Case V., p. 540).

the fingers and, perhaps, of the arms, may develop. There may also be a general trembling of the whole body. Hallopeau [1] in certain cases found convulsive tremor of the limbs manifested only at the end of expiratory movements, the rhythm of which reproduced it. There may also be seen peculiar contractions of the muscles of the face which go on continuously for some time. In other cases there are rhythmic twitchings of the eye-balls.

Leudet [2] reported the following case of partial chorea following poisoning by charcoal fumes. A man, 61 years of age, attempted suicide. He was found at midnight, unconscious and struggling violently. Taken to hospital next day he was still unconscious, showing great restlessness and continuous movements of flexion and extension of the right arm, the strength of which movements was quite marked. There was also diminished sensibility of the skin. After four days intelligence had only partially returned. He then remembered his name and where he lived, but was not able to give any explanation of his antecedents or of his present condition. There was persistence of the choreic movements of the right arm, but only when he was awake. There was no paralysis. At the end of six days these movements had greatly diminished, and at the end of three weeks had completely disappeared. In Cramer's [3] case, which ended fatally, there were on the day before the patient died uneasy movements of the legs and arms; later there developed regular movements of crossing the arms, simulating the action of embracing.

In a case described by Trenel,[4] in which there were marked mental disturbances, choreic movements were from the first a prominent feature, the patient being very restless, making grimaces, and constantly moving her hands and fingers. These choreic movements became more intense, the irregular movements of the arms, and the grimacing face being quite characteristic. Sometimes when standing, the patient appeared as if she were being violently forced backwards or forwards; walking became very difficult, owing to the irregular movements of the legs causing her to trip herself; the fingers were flexed and extended in a spasmodic fashion; there were painful cramps in the muscles; and sometimes the movements in the muscles resembled those of tetany.

[1] Hallopeau: *Le Bulletin Médical.* Paris, 13th December, 1893.

[2] Leudet: *Arch. Génér. de Méd.* 1865, p. 523.

[3] Cramer: "Anatomischer Befund im Gehirn bei einer Kohlenoxydgasvergiftung. *Centralblatt für Allgem. Path. u. Path. Anat.*" Jena., 1st July, 1891, p. 545.

[4] Trenel: "De quelques symptomes consécutifs a l'intoxication aiguë par l'oxyde de carbone," *Gazette hebd. de Méd,* 1895.

Multiple Sclerosis following CO Poisoning.

So far as we have been able to discover, there are only two cases on record of multiple sclerosis following CO poisoning. The first was reported by Becker.[1] A man of 47, free from any disposition to nervous disease, who had never had syphilis, and who had been very healthy, sober, and industrious, was exposed to a powerful jet of illuminant-gas in a closed room. He immediately lost consciousness, and ten minutes elapsed before he was rescued. After two hours' artificial respiration he began to breathe spontaneously, and developed fine tremors in the muscles of the left side. After a few hours these became much more exaggerated and generalised, the least irritation bringing on intense muscular spasms. At the end of eight hours the convulsive movements became so violent that two men were required to look after him. The skin was very hot, and numerous red spots developed over the surface. The urine contained albumen but no sugar. The patient took no nourishment, for it was impossible to touch him without provoking convulsions. His pupils were dilated and sluggish. At the end of two days convulsions ceased on the left side. On the fourth day the patient was able to swallow liquids, but there was no reaction of the pupils; the temperature was 35·4°C. On the eighth day the convulsions had entirely disappeared, but profound helplessness, very marked stupor, and left hemiparesis lasted for eight days. For a considerable time he complained much of flashes of light, with vertigo and impaired vision. He slowly got better, although his intelligence still remained dull; there was still the drawling speech and a tendency to scan the words; he also suffered from tremors of the hands.

Three weeks after the accident he returned home, but in a short time he came back to the hospital, presenting some of the symptoms of multiple sclerosis, such as tremor of the upper limbs, which came on with voluntary movements, absence of tremor when at rest, tremor of the tongue when protruded, and tremor of the lower limbs. There was exaggeration of reflexes, especially on the right side. There was no tremor of the head, nystagmus, nor ocular disturbances. Becker regarded as the most significant symptom in this case the tremors, which was typical intention-tremor. During the patient's first stay in hospital immediately after the poisoning, the tremor appeared as he lay in bed, but Becker thought that this might be caused by mental excitement, since it appeared after he had been fatigued, as, for example, after minute clinical examination. As he became used to his surroundings, he learned to make voluntary movements more easily. There were marked changes

[1] Becker: "Ueber Nachkrankheiten der Kohlenoxydgasvergiftung, speciell über einen unter dem Bilde der Multiplen disseminirten sclerose des Centralnervensystems verlaufenen Fall." *Deutsche Med. Wochen.*, 4th July, 1889, p. 540.

in his speech to which attention has already been drawn. In this case, as in so many cases of CO poisoning, there was defective memory, a considerable portion of the time following his exposure to the gas being erased from his mind. At the end of five years all the symptoms, the tremor, the scanning speech, the intentional tremor, the shaking of the hands when writing, and the staggering gait and giddiness, had become worse.

In the second case, that of Etienne, which was recorded by Avramoff,[1] a man of thirty years was in July 1894 exposed to fumes from burning charcoal, and was rescued in an unconscious condition by his companions. He appeared so little affected that in a day or two he was able to resume work. But eight days later he began to complain of tingling in the left hand, then in the right, though always more marked in the left. His condition quickly got worse, his legs trembled when he stood, and he had difficulty in walking and stooping. Intentional tremor began to be distinct, more especially in the left hand. Speech was tremulous, and on different occasions it became almost stuttering. The following year tremor of the head started, and two years after the accident, during which time there had been distinct intervals of improvement, the spasmodic movements got worse.

In 1897, the following was his condition :—Whenever he attempted to stand, he swayed backwards and forwards for a little before finding his equilibrium. In walking he had difficulty in raising his feet from the ground, and he lifted the leg all of a piece, replacing the foot toe first on the ground with a good deal of faltering and trembling. He could stand fairly well even on one foot when his eyes were shut. When asked to perform certain movements with his arms or with his legs when lying in bed, the tremor started at once, and he had the greatest difficulty in executing them. Generally the movement ceased during sleep, but under certain influences, as cold, the trembling appeared. There was no tremor of the tongue when protruded; no muscular atrophy; but there was fibrillary tremor in the thigh muscles. Intelligence was unaffected, and generally the memory was good, unlike the previous case. The patient sometimes complained that his head was heavy, and sometimes that he had headache. Horizontal nystagmus was present. His condition became worse, and he walked only with the greatest difficulty; the intentional tremor increased in intensity; the reflexes became greatly exaggerated; and the speech was now markedly scanning.

Regarding the causation of this condition, Becker believed that CO poisoning could produce, like infectious and other toxic conditions sclerotic changes in the brain and spinal cord, these changes being the result of multiple hæmorrhages and softenings due to changes in the walls of the blood vessels.

[1] Avramoff: "Contribution à l'Étude des Affections Nerveuses Chroniques consécutives aux intoxications aiguës." *Thèse de Nancy*, 1900, p. 37.

CHAPTER X.

DISTURBANCES OF THE CENTRAL NERVOUS SYSTEM.

Neuritis, Paralysis, and other Nervous Disorders following CO Poisoning.

Under the heading of CO poisoning in mines, attention has been drawn to the fact that nervous complications such as paralysis, including hemiplegia and paraplegia, are occasionally met with, as well as local disturbances in the motor, sensory, and vaso-motor nerves independent of the central cerebro-spinal system. These cases are, however, much more frequently met with in poisoning by charcoal fumes, and in furnace, producer, and illuminant gases, where the percentage of CO is much greater.

On this question much has been written by German and French writers, but in our own country, judging from the Report of the Departmental Committee on Compensation for Industrial Diseases, very little appears to be known. Only four cases were brought before the notice of that Committee; two by Sir Thomas Oliver and two by Dr Judson Bury. As a result, the Committee remarks: "Had a gas which is so common as carbonic oxide possessed noxious qualities of this character, we should have expected that the fact would have been recognised long since, and in many more than four cases." These remarks were made regarding chronic exposure to this gas.[1] But we are of opinion, and in this we are supported by Lancereaux,[2] Levy, Brouardel, and numerous French and German writers, that these nerve lesions are very much more common than reports seem to show, owing to the fact that it is not recognised by medical practitioners that neuritis may follow exposure to CO gas.

We have a case before us at the present moment in which a man, who was engaged cleaning the tubes in connection with a blast-furnace and ammonia plant, and who was constantly exposed to gas—containing on an average 25 per cent. of CO—suffered on several occasions from severe lightning pains down the back of the right leg, and considerable loss of power in the limb, with other symptoms. He found that when he was engaged in other work this condition disappeared in a short time, to

[1] See also the evidence of Professor Glaister in *Report of Departmental Committee on Industrial Diseases*, 1907, p. 142.

[2] Lancereaux: Quoted in Simon's *Thèse de Paris*, 1883, p. 68.

return, however, after he had been exposed for some time to the gas. Lancereaux frequently found cases of neuralgia and nervousness, certain intellectual disorders such as confusion of mind, hallucinations of sight, and partial loss of memory, and one case of paralysis with œdema, in cooks and ironers, who were constantly exposed during their work to fumes containing CO. Workers themselves generally lay the blame for the production of the symptoms on the wrong thing—*e.g.*, exposure to cold after coming out of a superheated room. Simpson[1] writing on poisoning by CO, says "Some years ago I used to see an 'ironer' who when she awoke in the morning was the subject of vertigo, headache, and visual hallucinations, but had no amnesia or sickness. She was always worse after "glossing" and during drying. She stated that many other women in the trade suffered from headaches, etc., but they usually attributed it to the heat." The only British authors who have attempted any detailed study of this condition are Ross and Bury in their work on Peripheral Neuritis.

Portal, in 1775, in his "Observations sur les effects de vapeurs mephitiques sur le corps de l'homme," was the first to describe cases of paralysis, one with trophic disturbances, following CO poisoning. A young woman, after asphyxiation, remained five days unconscious; an extensive ecchymosis appeared on the side on which she had been lying, and for many days it was impossible for her to walk, while she also felt considerable numbness in the lower limbs. He also described the case of a young man, 21 years of age, who after poisoning by charcoal fumes, showed great impairment of memory, and who suffered also from paresis of all his limbs. His legs were so weak that they could not sustain his weight. He gradually regained his strength, but memory was slower in returning. But even before Portal's time Boerhaave had written that "vapor carbonum apoplexiam producit." In 1839, Golding Bird published a most interesting and searching clinical study of "Poisoning by the Vapours of Burning Charcoal and Coal," in which he described a case of paralysis. This article is also of interest in that it contains, perhaps, the first description of the appearance of punctiform hæmorrhages in the brain which are considered by some more recent authors to be characteristic of CO poisoning.

It was not, however, till 1843 that a proper study of these conditions appeared, when Bourdon demonstrated the existence of localised paralyses following poisoning by charcoal fumes. Tourdes in 1857 reported cases of paralysis and loss of sensation among workmen in furnaces where gas was used for certain metallurgical operations. Leudet, in 1865, made a much more detailed study of the effects of CO poisoning on peripheral nerves. A few of his cases are very interesting; indeed they may be

[1] J. Christian Simpson: "Poisoning by Carbonic Oxide," *Lancet*, 29th Sept. 1900.

regarded as classic cases, since they give a vivid picture of the symptoms produced and the various lesions found. In the following year Laroche published a thesis in which he collected all the known cases, besides adding a few interesting and unusual cases which had come under his own notice.

We have collected a very large number of cases from literature, so that the various symptoms may now be described in detail which arise from the action of CO on the nerve tissue. We shall refer briefly to illustrative cases. The signs and symptoms are very varied in character and by no means constant in their appearance; indeed, this is one of the striking features of the action of CO on the nervous system. In many cases the paralysis comes on suddenly; the patient on coming to consciousness finding one or more limbs affected. In these cases, the exposure to CO has usually been prolonged, and the patient has been unconscious for a considerable period. But in a case reported by Borsari,[1] where a man was poisoned by the fumes from a charcoal stove and developed paralysis of the trigeminal nerve, he was not rendered unconscious. In a remarkable case of Sibelius, to be described (p. 298), in which blindness, dementia, etc., followed CO poisoning, the boy was exposed to the gas for less than five minutes. It has to be repeated, moreover, that in a considerable number of cases, nervous lesions have become developed after repeated exposures to small quantities of CO. Thus a number of cases of paralysis developed among furnacemen in an Ironworks in the north of England, and it was found that these were caused by the bell at the top of the furnace being defective, thus allowing the poisonous gases to escape. These men were never sufficiently affected to be overcome to the extent of the onset of unconsciousness. In the same way, cases are found among foundry workers and steel workers, etc., as well as among cooks, laundry workers, ironers, and others.

Motor Disorders—Paresis, Paralysis, Paraplegia, Hemiplegia, Monoplegia, etc.

One marked feature in nearly all cases is the partial loss of power over the limbs, both upper and lower, the lower being, however, much more frequently affected than the upper. This powerlessness depends upon the action of the poison on the central nervous system, and is quite distinct from the paralyses which we shall describe later. It generally disappears in a few hours; but some patients complain of slight weakness lasting for a few days. In mild cases all that is complained of is a slight stiffness and weakness, or a fatigued feeling in the muscles, which quickly passes off. But paresis may be marked and last for weeks; in

[1] Borsari: *La Reforma Medica*, 8th March, 1889.

some cases general paresis, which lasted for a considerable time, has been noted. This loss of muscular power sometimes prevents the men escaping, although they understand perfectly well they are in danger. Many cases are on record where this powerlessness, attacking only the lower limbs and leaving the upper unaffected, has led to the patient's escape, by employing their hands, for example, in one case, to open a carriage where the heating apparatus had caused symptoms of poisoning, and in another to break a pane of glass with a stick when a room was full of gas. Again this powerlessness of the limbs may, where the man is working in a perilous position, lead to serious results; for example, a man who was gassed on the top of a gas producer fell to the ground, a distance of seven feet, and received a fracture of the base of the skull from which he died.

In some cases, owing to a sense of muscular weakness in the neck, the head is held far back and stiffly, the patient feeling that if he does not keep it in that position, his head will roll forward in a helpless condition.

Sometimes a curious gait is produced. The patient trails his feet as if dragging a weight or, owing to foot-drop, he requires to lift them higher than usual to prevent tripping, giving him a typical steppage-gait. Some of the men at the same time complain of tingling and prickling or a feeling "of pins and needles," or of a peculiar numb feeling in their legs. In most of such cases the symptoms disappear in about 24 hours. It is not only loss of power which is felt in the limbs; in some cases it is rather loss *of muscular co-ordination*; and the patient finds that he is not able to execute the movements he desires, although he fixes his attention intently on what he is trying to do. In some cases this muscular inco-ordination appears only when the patient is standing, as when lying in bed he may be able to execute many different movements. When there is this loss of co-ordination it may be discovered at the same time that the patient has lost confidence in himself. This may last many days; and in some rarer cases the patient has to learn to walk like a child. Even where there is considerable improvement, excessive fatigue may bring on a relapse, and the staggering gait may return. The same thing occurs with loss of power in the limbs, which may last for weeks or months. We have seen cases where the paresis persisted for months, coming on especially when the patient had walked too far. Sometimes the legs give way very suddenly and the patient falls to the ground.

Dr Motet, [1] in a most interesting description of his own symptoms after poisoning by CO, describes the peculiar action it had on his limbs. On the third day when he began to try to walk, he staggered about like

[1] Motet: *Annales d'Hygiène Publique*, 1894, Vol. XXXI., p. 261.

a drunken man; swayed towards the right side, was seized with great giddiness, and had to lie down again. On another attempt he found he could stand erect if he stiffened his limbs, but the least attempt at walking brought on the disordered movement. While lying in bed, however, he found he could quite easily move his limbs and put them into any position. The following day he again tried with the same result, viz.—as soon as he attempted to walk the power of muscular co-ordination disappeared, and he found that he could not balance himself properly. Indeed his attempts at walking were very like those of a child learning to walk. He required something to support him or he fell. At the end of eight days the difficulty in balancing himself remained. He developed a peculiar attitude, holding himself rigid with the head well back as if it were too heavy, and as if the muscles of the neck alone were not able to prevent it falling forwards. After fifteen days he could go downstairs holding firmly to the stair-rail, relying on the strength of his arms for support, but he still dreaded losing his balance. After six weeks he had not fully regained his powers of locomotion, for although the giddiness had disappeared, he still had fainting attacks. He had not yet regained firmness on his legs, and at times felt himself drawn to one side, his balance not having become normal.

The following is an interesting account by Glaisher[1] of this powerlessness developing in a very rarefied atmosphere at an altitude of 29,000 feet. He writes:—"Shortly after, I laid my arm on the table—possessed of its full vigour, but on being desirous of using it I found it powerless; it must have lost its power momentarily, for on trying to move the other arm I found it powerless too. Then I tried to shake myself and succeeded, but I seemed to have no limbs. On looking at the barometer my head fell over my left shoulder. I struggled and shook my body again, but could not move my arms; setting my head upright for an instant only, it fell on my right shoulder, then I fell backwards, my back resting against the side as in the case of the arms, so all muscular power was lost in an instant from my back and neck."

Where this powerlessness has been marked, it is often found that on recovering from it there are darting pains in the limbs, or a vague feeling of soreness of the muscles. There may also be disturbances of sensation of the skin.

Paralysis.

In other cases there may be actual paralysis. This may be found when the patient regains consciousness, or it may develop days or even weeks after the acute symptoms of poisoning have passed off. In such

[1] Glaisher: *Travels in the Air.*

cases the development may be sudden, the patient becoming paralysed in a certain part without any premonitory symptoms, or after an attack of severe pain or sensory disturbance in the part. Again, it may come on slowly, paresis passing into paralysis, or the patient may complain of vague pains in the affected limb, accompanied by gradual and increasing weakness and, in some cases, by sensory disturbances, such as formication or anæsthesia. In most of the cases, however, the patient makes little complaint, the loss of power itself being the only disturbing symptom. In the chronic variety, the signs of slow poisoning by CO, viz. :—headache, which may be severe, causing the patient temporarily to leave his work, and from which he quickly recovers in the open air; giddiness; a feeling of being easily tired; loss of appetite; and slight loss of control over the legs, with muscular weakness—may go unheeded, until one day the symptoms become more pressing, and the patient stops work with the appearance of the paralysis.

We have found that the following description by Ross and Bury[1] of the development of paralysis following constant exposure to CO is almost typical. For six months a man complained of shortness of breath, shooting pains in the legs, shoulders, and arms, numbness of the hands and feet, and severe cramps of the calves of the legs. His legs began to swell, and became so weak that he could hardly walk. He had the "high-stepping" gait caused by foot-drop from paralysis of the anterior muscles of the leg. He had no wrist-drop, but the power of extension was feeble. The face was expressionless and destitute of wrinkles. The toes were almost completely insensible to pain, and there was also considerable anæsthesia of the dorsum of the feet, but not of the legs. The plantar and popliteal nerves were painful on pressure. The patient, on closing his eyes, complained at one time of seeing faces and scenes like pictures, and he was much afflicted with sleeplessness and horrid dreams. There was enlargement of the area of cardiac dulness, the pulse was 90, regular, and very compressible, but there was no sugar or albumen in the urine. All the muscles reacted to a weak faradic current. Twelve months after leaving work he had made great improvement, the paralysed muscles having to a considerable extent regained their power.

Their second case is equally interesting. It was that of a furnaceman aged 43 years. Eighteen months before he began to complain of general weakness, his legs felt heavy and frequently gave way under him. For the last twelve months numbness and tingling of the hands and feet had developed, and there was giddiness while walking, as well as breathlessness. His appetite failed, he was troubled with sleeplessness, and his rest was broken by horrid dreams. He had a severe attack of diarrhœa, after which he had become much worse. On standing with his feet together

[1] Ross and Bury: *Peripheral Neuritis.*, p. 169.

he swayed. He had also high-stepping gait, owing to foot-drop. His grasp was feeble, and he had great difficulty in performing delicate movements with his hands. His face was expressionless and devoid of wrinkles, but there was no actual paralysis of the facial muscles. He had difficulty with his bladder. He complained also of tingling of the hands and numbness of the finger-tips, of burning sensations in the soles of the feet, and of a dead, numb feeling in the toes, but there were no marked sensory disorders to be made out. The cardiac area was somewhat enlarged, the pulse numbered 85, and it was regular but very compressible. There was no œdema of the ankles. On raising his hands suddenly, severe giddiness was induced. The urine was normal. Both these men complained first of pain about the hypochondriac region and shortness of breath on exertion. Both suffered from anæmia. In a case reported by Glynn[1], of chronic poisoning in a boy of 16, the first complaint was of pains in the calves of the legs, followed a week later by swelling and gradual loss of power in the lower limbs, the extensors of which were found to be paralysed.

Regarding the time of onset of paralysis after CO poisoning, the fact that it may appear weeks after the exposure is a most important point to remember in connection with the question of accident compensation.

From a careful study of the paralyses following CO poisoning, one characteristic feature, which is sometimes a considerable aid to diagnosis in chronic cases, stands out prominently, viz. :—the paralysis is most varied as to its seat and its type. There may be every variety from paralysis of one muscle or group of muscles to that of a limb (monoplegia), while a considerable number of cases of hemiplegia are also on record. We call attention to the fact that the right side of the body is more often affected than the left, also that there may be paralysis without any impairment of sensation in the affected part, and with no trophic lesions, while in some cases the two latter conditions may be encountered without paralysis. As in the case of alcoholic neuritis, which we shall have occasion to compare with and differentiate from CO poisoning, the extensor are more generally attacked than the flexor groups of muscles.

Cases are on record by Bourdon, Rendu, Jacoby, and others, in which the paralysis produced was not unlike that produced by lead poisoning. In Bourdon's[2] case, there was paralysis of the forearm and hand affecting the extensors of the fingers, and generally the muscles supplied by the musculo-spiral nerve. In his report no mention is made of the state of the supinator longus. In Jacoby's[3] case, a girl of eighteen, there was paralysis of the extensor communis digitorum, the extensor longus

[1] Glynn : *Brit. Med. Journal*, 6th April 1895.

[2] Bourdon: *Thèse de Paris*, 1843.

[3] Jacoby : Quoted by Vialettes. *Thèse de Paris*, 1895, p. 134.

proprius, and the abductors of the thumb, but the supinator longus was not implicated as is found in lead poisoning. There was slight disturbance of sensation in the area supplied by the musculo-spiral nerve.

It has already been remarked of some cases of generally distributed paralysis that one side recovered almost entirely in a day or two, leaving hemiplegia. This was found in the case by Comby to be described (p. 264), and the same is found in monoplegia. In a day or two the paralysis is found to be localised in one group of muscles. In other cases there may be ascending and progressive paralysis, the paralysis of the extensors of the legs being followed by paralysis of the other muscles, then later the arm is attacked and, perhaps, the face.

Leudet[1] relates the following case of a man who attempted suicide by inhaling charcoal fumes, and who suffered from incomplete loss of consciousness and a general state of numbness. On regaining consciousness, he complained of a dull, sometimes stabbing pain in the right buttock at the point where the sciatic nerve emerges. Over this there was a red patch of the size of the palm, the skin being a little raised and painful on pressure. The pain darted along the sciatic nerve. Thereafter a gradually-increasing weakness of the extensors of the right leg developed, which extended till the whole leg was absolutely paralysed. The upper limbs were then attacked, and the patient was hardly able to raise his hands to his face; then the muscles of the face were affected; deglutition became difficult; and a slight embarrassment in speech developed. Twenty-eight days after exposure to CO there was complete paralysis of both upper and lower limbs, speech and swallowing had become almost impossible, and there was pronounced facial paralysis of the left side. Delirium set in, and the patient died thirty days after exposure to the fumes.

A case was reported by Lancereaux[2] of a man forty-five years of age, who was poisoned by CO. Two months after his accident, he was brought into hospital complaining of general muscular weakness; indeed he could hardly move in bed. Closer examination showed that the right side was almost completely paralysed, and that the left side was very feeble. Memory and intelligence were fairly well preserved. He died. At the post-mortem examination no marked cerebral lesion was, however, found. Again, we may observe paralysis of the face supervening weeks after an attack of paralysis of the extensors of the lower limbs, as in a second case by Lancereaux.

Contractures are occasionally met with; for example, in a case recorded by Rendu, in which along with complete hemiplegia of the right

[1] Leudet: *Archives Générales de Médecine*, 1865, Vol. I., p. 521.

[2] Lancereaux: Quoted by Simon, *Thèse de Paris*, 1882, p. 5.

side with involvement of the orbicularis palpebrarum, there was permanent contraction of the little and ring fingers. In Vergely's[1] case, also, contractions were seen. A woman, aged 22 years, was poisoned by CO. The right leg and foot were found swollen, and firm pressure on the calf caused acute pain. At certain points of the leg and foot there was anæsthesia. Pricking with a pin was felt in the leg, but not in the foot. Deep pricking was painful in the posterior part of the calf, but in other parts did not give even the sensation of contact. The patient felt her foot with difficulty, it had a disagreeable sensation of heaviness, and seemed as if it were squeezed in a vice. The plantar reflex was abolished in the right foot, but the patellar reflex was normal. There was considerable pain in the leg and in the foot, that in the leg being of a shooting character. The posterior part of the right thigh was swollen. The movements of flexion of the leg and of the foot and toes were very limited and painful. In course of time *the right leg became flexed almost at a right angle on the thigh.* The tendons of the biceps, sartorius, and semitendinosus muscles were rigid and tightly stretched. Movements of extension were so limited that they could hardly be detected, the foot being fully extended on the leg, passive movements, however, causing no pain. The foot was markedly anæsthetic, and also the inferior two-thirds of the leg and its posterior part up to the middle of the popliteal space. At the level of the anæsthetic regions the patient perceived sensations of heat and cold. Pricking only caused a sensation of touching. As the swelling went down the patient complained even more of the pains in the leg. A month later anæsthesia was diminishing, but the leg was still strongly flexed at the hip joint.

Paraplegia.

We have already described cases of paraplegia following poisoning in pits, and two cases recorded by Portal. Laroche[2] reported the case of a woman, 35 years of age, who showed complete paraplegia following CO poisoning. After six weeks the left leg was slightly improved, the right leg still remaining absolutely paralysed. The feet were both strongly extended, and the big toes were flexed on the feet. At the end of two months the right leg showed signs of improvement. At the end of six months the left had completely recovered, and there was considerable improvement in the right. The big toe was still firmly flexed, and, as the patient walked about a good deal, the constant pressure produced a gangrenous patch on the toe. At the end of eight months the patient still swung her leg sideways when walking, but the improvement in the

[1] Vergely: Quoted in Vialettes' *Accidents consécutifs à l'Empoisonnement par l'oxyde de carbone*, 1895, p. 93.

[2] Laroche: *Thèse de Paris*, 1865, p. 28.

muscles had been continuous. In Briand's[1] case (p. 180) there was complete paralysis of the legs which lasted for several days. The patient slowly recovered the use of her legs, but at the end of a month there was a certain amount of anæsthesia which lasted for a considerable time. This paralysis Briand thought to be very similar to that observed in alcoholic intoxication. In Croizet's[2] case there was total loss of power of both legs and arms. At the end of six weeks the patient could move his left arm, then movement in the lower limbs began to be manifested, till at the end of two months the right arm could perform imperfect movements, although it was a long time before he could sustain himself on his legs sufficiently well to walk.

In other cases both upper limbs may be affected, the legs escaping altogether. This happened in a case cited by Gautier[3] where a man and woman were poisoned by the fumes from an American stove. The woman quickly recovered, but the man was seized with deafness, severe headache, and paralysis and œdema of both arms, which disappeared very slowly under treatment.

The following case, in which there was paraplegia, accompanied by marked trophic as well as mental disburbances, was believed by Panski,[4] who recorded it, to be one of disseminated sclerosis or acute encephalomyelitis. A cabman, 28 years of age, was found in an unconscious condition in his house along with his child who was dead. His wife, when disturbed by the neighbours knocking, was quite angry with them for disturbing her, and had no idea that her child was dead and that her husband lay unconscious. She recovered very quickly. The man was removed to hospital five days after the accident. Deep necrotic areas were found over the sacrum and buttocks near the trochanters. On the right calf and on the back of the left thigh several large blisters were found. He was quite unable to move the lower limbs, but the movements of the upper limbs were quite free. Sensibility of the skin of the legs and lower part of the abdomen up to the umbilicus was slightly decreased, light touch was not felt, and the patient was not able to localise sensation properly. Sensibility to pain and to temperature was also much disturbed over the whole body. There was no pain in the paralysed limbs, and the nerve trunks were not sensitive to pressure. Electrical reaction showed no change. He spoke very slowly and in a broken manner, and the syllables were

[1] Briand : *Annales d'Hygiène Publique, etc.*, 1889, Vol. XXI., p. 356.

[2] Croizet : *Thèse de Paris*, 1903.

[3] Gautier : *Bulletin de l'Academie de Médecine*, 1889, p. 172.

[4] Panski : "Ein Fall von Acuter disseminirter Myelitis oder Encephalomyelitis nach Kohlenoxydvergiftung mit Uebergang in Heilung." *Neurolog. Centralbl*, 1902, p. 242.

separated by longer or shorter intervals. There was also dysarthria. The movements of the face and tongue were a little slower. Incontinence of urine and fæces gave considerable trouble. The patellar reflexes were very much increased, and ankle-clonus was well marked on the right but weaker on the left side. Cremasteric and abdominal reflexes were weak, but the plantar were increased.

His mental condition was interesting. He was very dull, and when questioned he did not appear to comprehend or understand, for his answers to reiterated questions on the same point were often different and always wrong. While he was being examined he would stare into vacancy, and would often fall asleep. Everything, from the time he came home on the night of the accident till nine days after, had been erased from his memory. He listened to the details of his wife's illness and his child's death with indifference. He had no idea of what was wrong with himself, or how his accident had been caused. He also became melancholic. After a fortnight sensibility had returned to normal, and he was quite conscious of his condition. A month after the accident he was able to make slight movements with his legs, but in doing so there were clonic convulsions in whole groups of muscles. There was no degeneration of the muscles. The disturbance of speech had now almost disappeared. A fortnight later he could stand without being supported; when walking he displayed the typical spastic gait. The bladder disturbance existed only during sleep. After three months in hospital the sensibility of the skin to touch, pain, and temperature was normal; reflexes were now good, the patellar being still increased, while ankle-clonus could still be demonstrated. He was now quite able to to walk, but was easily tired. Speech was quite distinct but a little slow, and his mental condition was still dulled. Two months after leaving hospital, very little could be found wrong with the patient except increased reflexes and slight weakness of intelligence. He returned to work but had no energy, either physical or mental, and no initiative. From the rapid progress of the disease, the principal symptoms of which were spastic not degenerative paralysis of the lower limbs, incontinence of urine and fæces, vaso-motor and trophic disturbances, somnolence lasting for days, dulled cerebration, slow, indistinct speech, loss of memory, etc., Panski regarded the disease as an acute encephalomyelitis or disseminated myelitis.

In some cases of paralysis of the lower limbs, careful examination shows that certain groups of muscles only are affected, and where this obtains it is generally the extensor group. Glynn[1] reports the case of a boy of 16 who was constantly exposed to CO at his work. He began to complain of pain in the calves of the legs, which was followed by

[1] Glynn: *Brit. Med. Journal*, 6th April, 1895.

complete loss of power in both limbs. On examination the extensors were found very weak; there were loss of reaction to the faradic current, tenderness of the muscles of the leg when squeezed, much impaired sensation, paræsthesia, severe spasms of the gastrocnemeii muscles, and loss of knee and plantar reflexes. Recovery was rapid, although the extensors still remained weak and the knee-jerks absent.

Hemiplegia.

There may be complete hemiplegia resembling what is exhibited in cerebral hæmorrhage, viz.:—paralysis of the arm and leg and part of the face on the same side. This may come on suddenly or may appear after a few days. Faure[1] reports a case where a man was found unconscious in bed asphyxiated by CO, and sensation was disordered for a day or two. Some days after recovery from this, the patient found that his right arm and leg had lost their feeling, and motor paralysis then developed. At the same time the face was pulled to the left side, the muscles being quite flaccid on the right. Laroche[2] also describes a case of hemiplegia which developed gradually. At first there was loss of sensation in the right hand and right leg, then paralysis developed in these parts, which spread till the limbs of the right side were absolutely powerless and flaccid. The right side of the face was next attacked, the muscles being quite flaccid and the eyes closed. Loss of sensation frequently precedes or ushers in the paralysis.

In Pérochaud's[3] case of hemiplegia, which was found on the patient regaining consciousness after being four days in a comatose condition, the face was not affected. Laroche[4] also quotes the case of a woman, 32 years of age, who was exposed to charcoal fumes during the night. She rose next morning in a state as if she had been intoxicated by alcohol. Two days afterwards she was suddenly seized by hemiplegia, the face, however, escaping. This disappeared in a few days.

In the remarkable poisoning case reported by Tourdes,[5] where five out of six people exposed to coal-gas died, the survivor was unconscious for a few hours. On recovering, she complained of pain in her cheek and in the right side of the head. She could move her left hand slightly, but her right arm and leg were completely paralysed, the muscles of the face being quite free. The arm recovered sooner than the leg. There was painful tingling in her right leg. After a month the paralyses were

[1] Faure: *Archives Générales de Médecine*, Vol. VII., 1856, p. 554.

[2] Laroche: *Thèse de Paris*, 1865.

[3] Pérochaud: *Gazette Médicale de Nantes*, Sept. 1894.

[4] Laroche: *Thèse de Paris*, 1865.

[5] Tourdes: *Relation médico-legale des Asphyxies occasionnées à Strasbourg par le gaz d'éclairage*, Paris, 1847, p. 58.

greatly diminished. There was slight loss of sensation in the affected limbs which quickly disappeared, but as it disappeared considerable pain developed. In some of the cases of hemiplegia, as Comby's[1] (see p. 264), it is found, when the patient regains consciousness, that there is general paralysis, but that in the course of a few days movement returns very freely to one side, the other side remaining paralysed, perhaps for months. In some of the cases of so-called hemiplegia we find that it is only groups of muscles in the arm and leg which are attacked, thus occasioning what might be termed incomplete hemiplegia. Rendu[2] reported the following case, where a woman of 31 years was poisoned by CO and rendered comatose for twelve hours. The right side of the body and right side of the face were found paralysed, but there were no mental symptoms nor derangements of special nerves. Close examination showed that in the upper limb there was paralysis of the muscles supplied by the musculo-spiral nerve, loss of power in extensors of lower limb, and diminution in power of the flexors, but that the supinator longus was unaffected. In the leg the extensors of the foot only were affected, the muscles of the calf being normal. There was also loss of sensation in the lower part of the forearm and of the left leg, and in the neighbourhood of the little finger and inner surface of ring finger. Electrical contractility was lost in these parts for a month. Profuse perspiration was present in the anæsthetic areas, and there was also œdema of the right arm. Landouzy[3] reported a case of right hemiplegia where localised swellings developed over the biceps, back of neck, thigh, and foot, with anæsthesia in these parts. The skin in other parts showed patches of erythema. Laroche[4] reports another case of a young girl of 20 in whom there was hemiplegic paralysis of the left arm and leg, and of the left side of the face, the speech being much affected and the face exhibiting an imbecile look. The patient complained for many months of feebleness in the affected parts, but gradually recovered.

One of the most detailed descriptions of hemiplegia following CO poisoning is that recorded by Lereboullet and Allard.[5] As it presents several interesting features, we shall give the facts in detail. This case, we should add, has been quoted by several authors as a good example of pseudo-tabes following exposure to CO. A man, 44 years of age, being in misery and want, attempted suicide by inhaling fumes from burning charcoal. On 19th March 1895, he was removed to hospital in a condition of deep coma. His limbs were rigid and contracted.

[1] Comby : *Archives Générales de Médecine*, Vol. CXLIV., 1879, p. 513.
[2] Rendu : *Societe Médicale des Hôpitaux*, 13th Jan. 1882.
[3] Landouzy : Quoted by Vialettes, *Thèse de Paris*, 1895, p. 135.
[4] Laroche : *Thèse de Paris*, 1865.
[5] Lereboullet and Allard : *Revue Neurol*, 1899.

Next day he was very much better, but it was found that he had complete hemiplegia of the right side, the muscles of that side being quite flaccid and insensible to any stimulation. Patellar reflexes were present, but the right was feebler than the left. The face was relatively little attacked. Although he made great efforts to speak, he was quite unable to emit a sound. He had incontinence of the sphincters for a few days. Three days after the incident he began to speak a little. There was no history of alcoholism, syphilis, or neurosis, and his family history was good. On the fifth day after the attack, œdema of the right arm appeared, being most marked on the dorsum of the hand, and becoming less as it ascended till it stopped a little above the bend of the elbow. It pitted deeply on pressure. There was complete loss of sensation from the shoulder to the fingers; above the point of the shoulder there was a zone of hyperæsthesia; and thermo-anæsthesia, and analgesia appeared to extend three inches further up than the loss of touch. There was also an elongated eschar behind the shoulder. The œdema and this eschar gradually disappeared, but the arm remained powerless and anæsthetic. All the movements of the arm, except slight abduction and elevation, owing to partial preservation of the deltoid, were abolished. In a very short time muscular atrophy, especially of the triceps, was found to have developed. In the foot there was characteristic foot-drop. The patient complained of intense pain in the foot, the mere contact with the bed-clothing being sufficient to provoke it, and light pressure or passive movement of the foot made the pain almost unbearable. Intense pain was also caused when the muscles of the calf were squeezed, and when pressure was exerted over the sciatic nerve or its branches. Touching lightly and pinching the dorsal surface of the foot and lower two-thirds of the leg also caused severe pain. At no point in the lower limb was there anæsthesia; there was no trophic disturbance and but little muscular atrophy. The electrical reactions in this case will be discussed later.

A family, consisting of father, brother, and two sisters, were poisoned by CO. One of the sisters was found dead, the other was unconscious, and remained in that state for three days. On recovering, she found she could not move her left arm and leg. On examination, Wolff[1] found that she could only lift the arm a very little, and that she could not bend the fingers. The skin was so tense that if the fingers were forcibly bent she complained of great pain. She walked only with the greatest difficulty owing to the pain in her foot. The hand and foot were swollen. Areas of anæsthesia were present, these being irregularly distributed about the ankle, lower third of the leg, and over the wrist. Movement gradually came back, the fingers being the last to recover.

[1] Wolff: *Inaug. Dissert.* Greifswald, 1899.

Cases of hemiplegia are reported by Bourdon, Simon, Poelchen, and others. The following case, the details of which were reported by Revenstorf,[1] ended fatally. A woman, 46 years of age, was rendered unconscious by exposure to CO for a lengthened period. When found, there was spasm of all the muscles of the body. At the end of two days she became conscious, when it was discovered that there was hemiplegia of the right side with paralysis of the right side of the face. The patient died after a few days' illness. Death was produced by a considerable cerebral hæmorrhage. Such cases as have been described by Poelchen, Simon, Sibelius, and others, in which considerable areas of softening in the brain substance were found, generally end fatally after a comparatively short interval. We are able, then, to differentiate cases of hemiplegia of central origin from hemiplegia due to neuritis and, therefore, peripheral in origin, as, for example, in the case of Pérochaud and of others we have quoted. In the latter cases recovery generally takes place. But there is generally a fatal termination in cases with gross cerebral lesions, such as large cerebral hæmorrhages, found at post-mortem examinations in cases of illuminant-gas poisoning, and which have often led juries to bring in a verdict of death by apoplexy accelerated by gas poisoning.

Monoplegia.

In monoplegia it is generally the upper arm which is affected, although in some cases it is the leg. In most of these cases, one finds that a single group of muscles only is attacked, and that, later, paralysis gradually spreads till the whole limb is affected. Bourdon[2] reported the case of a strong woman of 30 years, who was poisoned by charcoal fumes, and who developed convulsions in which she remained for several hours. On regaining consciousness it was found she could not speak, there being pronounced deafness and, also, complete paralysis of the left arm. The following day her speech returned. Eleven days afterwards it was found that the left upper arm was completely deprived of power of movement, but that she could slightly flex the forearm; "sensation of the part was neither diminished nor increased; no pain; no swelling; no change in the colour of the skin. It showed the same temperature as the opposite side, but the muscles were slightly softer, more flaccid." She recovered completely in three months. This case is most unusual in that some sensory or trophic disturbance in the affected parts is commonly found.

Litten[3] reported the case of a man poisoned by gas, who was

[1] Revenstorf: "Fall von Hemiplegia nach Kohlendunstvergiftung. *Muenchener Med. Woch.*, 28th April 1903, p. 757.

[2] Bourdon: *Thèse de Paris*, 1843.

[3] Litten: *Deutsche Med. Wochenschrift*, 1889, Vol. XV., p. 5.

unconscious for a few hours. Twenty hours after he regained consciousness he developed complete motor and sensory paralysis of the right arm, with a hard brawny swelling from the tips of the fingers to the shoulder. The reflexes and electrical reactions were quite lost. Bullæ developed on the back of the hand. As feeling came back considerable pain developed. Litten held that the condition was the result of hæmorrhagic infiltration of the brachial plexus. A case similar to this has been reported by Klebs.[1] A man, 33 years of age, poisoned by charcoal fumes, awoke two days afterwards to find his right arm quite paralysed, and with complete loss of sensation; there was also marked swelling from the shoulder to the fingers; this infiltration was described as being like leather; pain was absent. A week after the accident reddish spots, on which blisters afterwards developed, appeared all over the body. Three weeks later the œdema became greatly less, disappearing altogether from the forearm. A large septic wound, however, was formed in the right axilla, marked septicæmia set in with considerable hæmorrhages from the wound, and the patient died a month after the accident. Alberti[2] reported a case where, after prolonged coma, the right leg was found completely paralysed and the foot everted and devoid of sensation. The patient also complained of stiffness of the back of the neck. In course of time extensive gangrene of the muscles of the neck developed, this being so deep that the right occipital protuberance and the right mastoid process were exposed, as well as the spinous processes of the third to the sixth cervical vertebræ.

Partial Paralysis; Paralysis of Groups of Muscles; Paralysis of Single Muscles.

Although when a patient regains conciousness it is frequently found that a limb is paralysed, yet in a day or two this disappears considerably, becoming localised in a group of muscles or, in rare cases, in a single muscle. Schachmann[3] describes the case of a girl of twenty whose left arm was found to be paralysed. Careful examination showed that the extensors were paralysed, the fingers were flexed *en griffe*, pronation was possible, supination slightly affected; indeed, all movements were possible save extension; tactile sensation and reflex sensibility were untouched. She was practically well at the end of ten days.

We have already referred to paralysis of the forearm affecting the muscles supplied by the musculo-spiral nerve, and where the paralysis simulated that produced by lead poisoning. In Dosseur's[4] case there

[1] Klebs: *Virchow's Archiv.* Bd. XXXII., 1865, p. 507.
[2] Alberti: *Deutsche Zeitschrift für Chirurgie*, 1884, p. 476.
[3] Schachmann: *France Méd.*, 1st July, 1861.
[4] Le Dosseur: *Thèse de Paris*, 1901.

was a most unusual type of paralysis of the Duchenne-Erb group of muscles, the deltoid, brachialis anticus, biceps, and supinator longus. In this case there were no trophic disturbances. In Leudet's[1] case, to which we shall refer in detail later, the paralysis was limited to the last three digits of the right hand, these fingers being markedly flexed. As the extensor muscles of the upper limb are most commonly affected, so is it with the lower limb. We have already described several cases of this where in both legs there was paralysis of the extensors. Jacoby[2] reports the case of a man of 38, who, after poisoning, suffered from feebleness in the right leg. Eight days after, paralysis of the tibialis anticus, extensor of the great toe, extensor communis, the peroneals and the interossei muscles was found, together with anæsthesia of the dorsum of the foot and of the outer surface of the leg. Afterwards the muscles atrophied. The patient recovered at the end of a month. Bloch[3] reported a case of peroneal paralysis in a girl of 15. There was at first foot-drop in both legs owing to paralysis of the peroneal muscles. This quickly disappeared from the right, but in the left the foot was inverted, and there was marked foot-drop. Eight days after the accident walking was still difficult, as the left foot dragged in a helpless manner on the ground. At the end of three weeks, however, there was considerable improvement.

In the following case reported by Maczkowski[4], there was both motor and sensory paralysis in the areas supplied by the posterior tibial and peroneal nerves of the left leg. Twenty-four hours after the man was poisoned by gas, a distinct increase in the size of the left leg and an ecchymotic discoloration of the posterior surface of the thigh were detected. There was also total analgesia in the left foot, a deadened feeling in the lower third of the leg, and anæsthesia of the inner surface of the left large toe. Hyperæsthesia was present in the area supplied by the obturator nerve, and there was weakness of the adductor muscles. Electric irritability was diminished, but there was no reaction of degeneration. The pain in the affected limb was so intense that walking was impossible. The patellar reflex was increased, but the plantar was absent. Œdema was present in the upper part of the thigh. After a fortnight there was great improvement, but the patient still complained of great pain, more especially in the sole of the foot and in the outer surface of the lower part of the thigh. Five months later sensation was normal, but the movements of the toes and of the foot were still weak, and there

[1] Leudet: *Archives Générales de Méd.*, 1865, p. 516.

[2] Jacoby: Quoted by Vialettes, *Thèse de Paris*, 1895, p. 134.

[3] Bloch: *Inaug. Dissert.*, Leipzig, 1902.

[4] Maczkowski: "Ueber Neuritis als folge der Kohlenoxydvergiftung." *Gazeta Lekarska*, 1899, Nos. 48-49.

was slight atrophy of the muscles of the leg. In another of Maczkowski's cases there was paralysis of the right anterior tibial nerve. The patient complained of severe pain in the leg, and there was an œdematous swelling of the foot and lower part of the thigh. From the beginning there was disturbance of sensation in the area supplied by the nerve, and this soon developed into total anæsthesia. There was foot-drop and paralysis of the tibialis anticus, extensor communis digitorum, and extensor proprius hallucis muscles.

One feature very characteristic of the cases of neuritis which follow CO poisoning, and which is a valuable aid to diagnosis, is the widespread and asymmetrical character of the nerve lesions, many different nerves being affected. The two following cases will serve as illustrations. The first is a case described by Schwabe[1]. It is very interesting from the ocular disturbances met with (see p. 267). A man, 36 years of age, was poisoned by coke-oven gas. He was brought home in a state of acute delirium which later developed into coma, in which condition he remained for a week. From the first the right leg was very much swollen, but this quickly disappeared except from the foot, which remained swollen and cyanotic. When he regained consciousness he complained bitterly of pain in the right arm and leg, and of a feeling of powerlessness and cold in the legs, more especially the right. The inferior branches of the left facial nerve were also involved, and there was optic neuritis, more marked in the left eye, with paralysis of accommodation. After a time there was considerable improvement in the left arm and leg, but the muscles of the right side became much relaxed, the right foot remained swollen and cyanotic, and he still complained of a sensation of cold and of great weakness in the lower part of the right leg from the knee downwards. Owing to the weakness and disturbance of sensation in the legs, walking was both painful and difficult. Improvement in this case was remarkably slow, the disturbances of sensation, contrary to what is generally found in such cases, taking a long time to disappear.

The second is one of the interesting cases reported by Maczkowski.[2] A young policeman was found unconscious, with swellings over the anterior aspect of the right forearm and the gluteal region. After a few days it was found that there were motor and sensory disturbances in the areas supplied by the left ulnar, radial, median, musculo-cutaneous, and middle cutaneous nerves. There was partial reaction of degeneration in the muscles of the left lower limb, and distinct motor and sensory disturbances in the areas supplied by the great sciatic, gluteal, cutaneous,

[1] Schwabe: "Ein fall von multipler Neuritis nach Kohlenoxydvergiftung mit Betheiligung der Sehnerven," *Muench. Med. Woch.*, 1901, p. 1530.

[2] Maczkowski: "Ueber Neuritis als folge der Kohlenoxydvergiftung," *Gazeta Lekarska*, 1899, No. 49: ref. in *Neur. Centralbl.*, 1900, p. 520.

anterior crural, small sciatic, tibial, and peroneal nerves. Severe pain was complained of in the sole of the left foot, and also in the lower part of the left thigh. After four months the condition of the upper arm improved very much, but that of the lower showed very little change. The reaction of degeneration was present in the muscles supplied by the anterior tibial and peroneal nerves, these nerves being the last to recover.

Boulloche[1] reported the following case of paralysis of the deltoid which developed in a man, 31 years of age, who was comatose for an hour after CO poisoning. On regaining consciousness, it was found that the patient could not raise his left arm to his head, or even move it. On examination, the movements of abduction and elevation of the arm were found to have disappeared, the shoulder curve was lost, being flattened by considerable atrophy of the deltoid. Sensation was unaffected, but electrical reactions showed marked degenerative changes in the muscle.

Reference has already been made to various cases of paralysis of the bladder. We shall deal later with Knapp's interesting case of paralysis of the muscles of the eyeball. A large number of cases of *facial paralysis* following CO poisoning have been reported. Some have been noted previously in these pages. In the Factory Reports, there is a case where double facial paralysis developed in a man who was gassed while repairing a kiln, which was situated between two others which were in use. At first he had difficulty in shutting his eyes. He also developed intense formication in the arms and hands, and inability to walk. Cyanosis and contracted pupils were also present, and the patient complained of great sleeplessness. This man was never rendered unconscious, although he must have inhaled a considerable amount of CO.

Disturbances of Sensation.

Immediately following acute poisoning we find general anæsthesia or, in other cases, hyperæsthesia, but the latter generally does not last long. As there is no single or uniform clinical type in the distribution of the paralyses following CO poisoning, but rather an infinite variety, so is it with regard to sensory disturbances. As in paralysis the loss of power may be the sole disorder, being in a certain number of cases unaccompanied by any sensory or vaso-motor disturbances, so sensory disturbances with no accompanying motor and trophic disorders may be the only indication which points to nerve lesions. But we find sensory disturbances much more frequently accompanying paralysis, and often with some trophic lesion. In other cases, although these other disorders are present, the disturbance of sensation is predominant. Disturbance of sen-

[1] Boulloche: "Des Paralysies consécutives à l'empoisonnement par la vapeur de charbon," *Archiv. de Neur.*, 1890, p. 212.

sation in many cases precedes the loss of power; it may be said, in a manner, to usher in the latter: and in some cases of paralysis, where there are at first no sensory disturbances, these may appear later in the illness. Where there are disturbances of sensation, there are also vaso-motor disturbances in a considerable number of the cases.

By far the most common sensory disturbance is *anæsthesia*. This may be general, as in the case recorded by Laroche.[1] A man of 50, poisoned by CO, was comatose for a day. Two days afterwards he was troubled with indistinct vision, seeing everything through a mist, and also with ringing in the ears. This lasted for three days. It was then found that there was complete loss of sensation all over the body, except in the front and upper part of the chest, neck, face, and scalp, even deep pricking with a needle not being perceived. Movement was unaffected. Four days later this condition continued, painful applications of all kinds causing no pain, even a violent blow on the thigh producing the same effect as a mere touch would ordinarily have done. The patient could walk, but could not feel the ground under his feet. Then prickling sensations developed in the shoulder, back, and over the abdomen, the anæsthesia gradually diminishing until seven days after the accident, when only slight evidence of it remained.

Gauchet[2] also describes a case where there was, with the exception of the head, neck, and breast, complete anæsthesia of the skin, which lasted for many days. The patient could walk perfectly, but did not feel the ground under him.

We have already pointed out that in serious cases of poisoning there is often complete anæsthesia of the skin, and when recovery takes place, that sensation returns first to the upper part of the chest from which it was the last to leave. Generally this anæsthesia quickly disappears. In some cases it may remain localised in a part of the body, in some instances the entire half of the body, or a limb, or part of a limb, or in the area of skin supplied by a certain nerve. Anæsthesia may alternate with hyperæsthesia and other sensory disturbances, as in the following case cited by Croizet.[3] A man, seriously poisoned by gas, first recognised those around him only on the fifth day. Complete paralysis of the legs followed the accident. And at the end of six weeks he could move his left leg, and by the end of two months all his limbs, although only in a very imperfect manner. Pricking with a pin caused acute pain in one part of the arm, while in the rest, even when the pin was driven in deeply, it was not felt. The next day the anæsthetic region would be hyperæsthetic. Sensation to heat and cold and to pressure underwent the

[1] Laroche: *Thèse de Paris*, 1865, p. 9.
[2] Gauchet: *Lyon Médicale*, 1857, p. 19.
[3] Croizet: *Thèse de Paris*, 1903.

same variations. Herpes appeared on the right arm and shoulder and along the right intercostal nerve, of which he had recurring attacks.

In hemiplegia, anæsthesia may be extensive, being distributed over the affected side, but in some cases it is only found in patches. In other cases there may be no sensory disturbances. It has also been found that where the paralysis is confined to a single group of muscles, as, for example, the extensor group of the arm or leg, there are frequently no sensory disturbances.

In some cases of paralysis an area of anæsthesia may exist over the paralysed part, and another patch, perhaps, in some other part of the body, whereas in other cases there may be no sensory disturbance over the paralysed part, while it may be present in some other non-paralysed area. In some cases there may be perception of contact and of temperature, the patient being able to distinguish the hot and cold test-tube, but not able to feel a pin-prick. Again, a patient may distinguish cold but not heat in certain parts, or, again, a part which appears quite anæsthetic to touch, reacts to the hot test-tube or to deep pricking.

In Knecht's[1] case (p. 271), in which there was aphasia and certain mental derangements, both hands were swollen and cyanotic, and bluish-coloured swellings were found over the right malar bone, right knee, and over the upper part of the sternum. Large blisters developed over the lower parts of the arms following the course of the ulnar and radial nerves, and over the hands and the right knee. There were no motor or sensory disturbances except in the hands, in which from the beginning paræsthesia had been present, a feeling of irritation and itchiness existing in the left hand, and prickling sensations in the right. For several weeks the *stereognostic sense* disappeared, so that the hand was quite useless although the movements were normal. Later, this patient developed a right-sided hemiplegia with facial paralysis, from which she made a complete recovery.

In Rendu's[2] case, in which there was incomplete hemiplegia of the right side of the body and face, including the orbicularis palpebrarum, the patient complained of a painful puffiness situated over the inner and lower part of the upper arm; also of not feeling objects which she touched with the hand, nor the ground under the right foot. Over the swelling on the arm, the skin was violet-red in colour and extremely sensitive to the least pressure. Indeed, Rendu feared an abscess might form, but little by little the swelling disappeared, till there remained only an indurated patch slightly painful on pressure. Over the face sensation was normal. On the lower arm impressions of touch, temperature, and pain were perceived, although somewhat feebly; but

[1] Knecht: *Deutsche Med. Woch.*, 1904, p. 1242.

[2] Rendu: *Union Médical.* Paris, 1882, Nos. 33 and 34, pp. 386 and 397.

there was anæsthesia of the dorsal surface of the wrist and hand. In the lower part of the leg this was more marked, absolute anæsthesia commencing in the lower fourth of the limb, becoming more accentuated in the tibio-tarsal region, and reaching its maximum on the dorsal and plantar sides of the foot. The anæsthetic portions of the skin of the hand presented a glossy appearance, the skin being tightly stretched, and the cutaneous folds covered with sweat had disappeared.

The following is an interesting case by Borsari[1] of paralysis of the sensory portion of the trigeminal nerve following CO poisoning. The patient never lost consciousness, but complained of vertigo and headache. The first day he found that the right eye was a little red, lachrymated, and was less widely open than the left. During the course of about a month, anæsthesia of the right side of the face developed over all the area of distribution of the trigeminal nerve, except the region of the angle of the jaw bone and the masseteric region, which was accompanied for some days only by modifications of the thermal sensibility of the buccal mucous membrane. There was no paralysis of the masseter or of the buccinator muscles. There was slight paresis of convergence of the eye balls, and slight vertigo of a passing character when the patient rose, but no other symptoms. Another interesting case of paralysis of the same nerve was reported by Lancereaux which is described later (p. 252).

Pain.

As must have been noted from the cases quoted, pain is a very frequent symptom. In many cases, the patient before the onset of the paralysis complains of pains which are often shooting in character, in other cases of a dull aching type. These pains may be located in a joint or even in several joints, which, with occasional accompanying effusions, compel the question of arthritis to be raised. Neuralgic pains in various nerves, but more particularly in the sciatic and facial nerves, are common. The pain may follow the course of the nerve, pressure on which sometimes causes exquisite pain; for example, the plantar and popliteal nerves in Ross and Bury's case were very sensitive to pressure. Pressure on the sciatic nerve at its point of emergence may cause acute darting pains in the leg. Neuralgia, in short, is frequently complained of in chronic poisoning by CO. This may pass suddenly from one nerve to another; for example, the sciatic nerve may be first attacked, and when that disappears, it may develop, as in Lancereaux's case, in the fifth cranial. In Croizet's case there was neuralgia of the ophthalmic nerve, there being dull, aching pain with occasionally shooting, darting pains; there were also shooting pains in the arms attacking one arm after the other; and after a few months, constant pain persisted in one arm which

[1] Borsari: *La Riforma Medica,* 8th March, 1889.

the patient could not localise. There may be painful spasmodic twitchings of the muscles; and severe cramps often set in which are generally worse at night. Soreness and vague pains in the muscles, a number of which may be affected, may be observed, and in certain cases, as in Glynn's, there is great tenderness of the muscles when squeezed. Loss of the muscular sense may also be met with. In some cases, a part of a nerve, as the sciatic, has been found to be much enlarged, and to be exquisitely painful when touched.

The following case of neuralgia following CO poisoning is reported by Bourru.[1] A workman, who was exposed for some moments to fumes at an open furnace door, dropped unconscious. Two other men at the same time were seized with vertigo and headache, but the fresh air quickly put them right. The man who was rendered unconscious, after he had recovered somewhat, was taken to the hospital. He was very restless and suffered from great dyspnœa; he had also severe attacks of vomiting and epigastric pain. He only remained a day in the hospital, as he appeared to have completely recovered, and at once resumed his work. Ten days afterwards, however, he returned to the hospital complaining of indigestion, frequent attacks of vomiting, cough, spitting of blood, and breathlessness. His lungs were found congested. Two days after he had recovered from this, he complained of acute pain in the lumbar region, constant in character and made worse by pressure on the muscles, more especially when the pressure was applied over the lumbar spinous processes. This pain was more severe and more extended on the left side, where it ran along the course of the sciatic nerve from its point of emergence to the inferior third of the thigh. There was no appearance of any paralysis. Although pain in the back and leg still remained in some degree, he had sufficiently recovered to return to work, but he had been at work only a fortnight when he experienced great pain in the trigeminal nerve, especially in the ophthalmic branch. The next day he had attacks of acute pain all over the right side of the face and head, and, curiously, with its onset the sciatica completely disappeared. Next night the facial pain, which had been gradually getting less, disappeared altogether, but once more the sciatica reappeared, and ten days later it was worse than ever. Over the spinous processes of the fourth and fifth lumbar vertebræ there was a spot which was intensely painful on pressure. A fortnight later there was considerable improvement, only slight sciatica remaining; but some feebleness of the left leg still remained without anything, however, of the nature of muscular atrophy. For a month or two more the patient remained feeble and pale. This case is instructive, showing as it does the late appearance of neuritis after CO poisoning, and the

[1] Bourru: *Archiv. de Méd. Navale*, No. XXVII., 1877, p. 232.

consequent difficulty in certain cases of determining, without a history, what caused it; the manner in which the neuritis quickly changes from one nerve to another; the obstinate character of the lesions; and lastly, the fact that the patient had inhaled the gas only for a very short time.

Muscular Atrophy.

Muscular atrophy may be found at the commencement of the paralysis, when in severe cases it may attack other than the paralysed muscles, but in the great majority of cases it progresses along with the paralysis, and is only found in the affected muscles. Where paralysis has existed for any length of time, there is always some measure of atrophy of the muscles owing to disuse; but it is not altogether dependent on this, and must, in certain cases at all events, be regarded as a primary result of the CO poisoning on the nerves.

Vaso-Motor and Trophic Disturbances.

All the known trophic and vaso-motor disorders have been met with in CO poisoning, from simple erythematous patches, with perhaps later development of herpetic vesicles, to deep sloughing involving the muscles, opening the joints, and causing the death of the patient by septic poisoning. Just as paralysis, unaccompanied by either sensory or vaso-motor disorders, may be found, so may the latter appear without paralysis or disturbances of sensation. Indeed, it is very exceptional not to find, in paralysis which has lasted for any length of time, some vaso-motor disorders; and where all are present, it may be found that the trophic disturbances are quite distinct and separate from the motor and sensory.

Attention has been already drawn to some vaso-motor disturbances which have been found in various parts of the skin in the acute stage of poisoning, and generally where coma has been profound. This usually disappears in a very short time after the patient comes to consciousness, but it may remain for a few days, disappearing from one part, only to reappear, however, in another. Vaso-motor disorders, on the other hand, are much more commonly found to supervene a few days after the accident. Bright cherry-red spots distributed over the whole surface of the body have been described by Klebs, Marthen, and others, these appearing very shortly after the accident, in some cases, indeed, while the patient was still in a comatose condition. Erythematous patches, oval, circular, irregularly-shaped, or in streaks, may be found either scattered over the body surface or may appear localised along the course of a nerve, such as the sciatic or branches of the facial, and in the latter case, but sometimes also in the others, herpes not infrequently develops. These areas

of reddening are generally flat, but in some cases there may be well-defined, distinct swelling of the part, giving sometimes the idea of the formation of an abscess, although it never goes that length. These patches are generally hyperæsthetic, but in some cases they are quite numb, and a needle may be thrust deeply into them without the patient complaining, while in others sensation is normal. Occasionally the patches are of a dull bluish-red cyanosed appearance and very painful on pressure.

The case reported by Leudet [1] gives a clear picture of such patches and other vaso-motor disorders seen after CO poisoning. A man, 30 years of age, was found in an unconscious condition with deep coma, general insensibility to pricking, a line of redness along the posterior, outer, and middle part of each forearm following the course of the musculo-spiral nerve, but with no swelling of the subjacent cellular tissue, this redness being more marked on the right than on the left side. A similar patch was found on the right temple near the external margin of the orbit. The following day herpetic vesicles developed on the latter patch, the redness becoming less vivid. No vesicles had developed on the arms, but the redness had become more marked on the posterior part of the right forearm, while it had almost disappeared on the left. The patient did not complain of pain, but only of a sensation of numbness in the three last fingers of the right hand, which he could not extend, these remaining in a state of exaggerated flexion. The movements of the thumb and index finger were normal. Two days later, herpetic vesicles appeared on the right forearm, and an eschar, about the size of the palm of the hand, developed on the lower part of the sacrum. Ten days after the poisoning, herpetic vesicles developed on the posterior surface of the right thigh, a vertical band of these ranging over the buttock and down the thigh along the course of the sciatic nerve. There was a little local pain, but no formication or other sensory or motor disorders of the thigh or leg.

In some cases pemphigus may develop, the bullæ being generally found on a reddened base which is sometimes dull and angry-looking. These occasionally develop into ulcers which are most intractable to treatment. They may be found over segments of a paralysed limb or along a nerve, but in other cases, as in a case of Hasse (p. 253) to be described, they may be generalised. A number of cases are on record in which pemphigus developed over the buttock and along the course of the sciatic nerve. In certain of these cases which ended fatally, *post-mortem* examination showed neuritis of the sciatic nerve. In cases by Rendu and others, the sciatic nerve, just as it emerged from the pelvis and for four or five inches along the buttock, was so much swollen that

[1] Leudet : *Archiv. de Méd.*, 1865, p. 516.

it simulated a commencing abscess. Rokitansky,[1] in the case of a young girl who died on the ninth day following CO poisoning, found that about the fourth day bullæ of various sizes developed. Rendu[2] reports the case of a patient who, in twenty hours after CO poisoning, made a good recovery from unconsciousness, complaining only of pains in the soles of the feet, but on each of these there soon appeared a large bulla of the size of a crown. On opening these bullæ, reddish gelatinous fluid escaped. There were no sensory disturbances of the part.

The cases by Klebs and Litten, already described, exhibited marked œdema of the paralysed part, the swelling, unlike the usual form of œdema, being quite firm and unyielding, and over this area the skin was found discoloured in parts. These hard œdematous swellings which, we should add, are frequently observed in cases of peripheral neuritis produced by various causes, may be found in various regions of the body. When they appear in cases attacked by paralysis they may not affect the paralysed part, but they may appear on other parts of the body where there is no motor or sensory disturbance. Brouardel[3] reported the case of a girl in whom the only sensory disturbances were anæsthesia and analgesia in the left limb, motor disturbance being absent. On the right lumber region, where she complained of considerable pain, there was a hard, elongated, non-fluctuating swelling measuring about seven by four inches. The left calf was harder and more swollen than the right. These swellings did not have the characteristic appearance of bruises. Over the swollen areas the skin showed an erythematous redness. Gautier[4] described the case of a man who, after poisoning by CO, was seized with paralysis and œdema of both arms, which symptoms, however, gradually disappeared.

Lancereaux[5] found in a man poisoned by illuminant gas, tremblings, sensation of stiffening in both legs, considerable difficulty in walking, and œdema first in the right leg and soon after in the left. Next day the swelling was marked. *The œdema was slight in the foot but more marked in the leg and thigh, the right being less livid and swollen than the left.* In another case of gas poisoning recorded by the same author, the œdema did not develop till eight days after the accident. In a case by Marthen[6] of a girl of 22, there was considerable pain and œdema of the left foot and leg, more marked in the thigh, and the veins in the thigh were swollen and varicose, no paralysis being present. The

[1] Rokitansky: *Wiener Méd. Preusse*, 1889.
[2] Rendu: *Union Médicale, Paris*, 1882.
[3] Brouardel: *Bull. de l'Acad. de Méd.*, XXI., p. 462.
[4] Gautier: *Bulletin de l'Académie de Méd.*, 1889, p. 172.
[5] Lancereaux: *Bulletin de l'Acadèmie de Méd.*, 1889, t. 21, p. 163.
[6] Marthen: *Virchow's Archiv.*, 1894, Bd. 136, p. 540.

œdema in this case quickly disappeared. In some cases, as in that by Lancereaux, it is found that the temperature of the affected part is considerably increased. The veins over the affected part may be greatly swollen and stand out like cords, but they do not have the hard feeling peculiar to phlebitis.

The following case by Lancereaux[1] is probably one of the most interesting on record. A woman, 22 years of age, five days after CO poisoning, complained of violent pains in the left calf. On examination the leg was found to be swollen, hard, and painful, especially on pressure. The superficial veins were distended. The left knee was swollen and contained a little fluid. Small *purpuric* spots surrounding the roots of the hairs were seen on the anterior and internal surfaces of the left thigh and leg. The temperature of the left leg was distinctly increased. From the colouration and temperature the circulation appeared to be going on more energetically, as pressure on the skin made the red colour disappear but left little pitting. The distribution of the œdema was unusual, there being none about the malleoli or on the dorsal surface of the foot. It was only present over a small area of the tibia. There was no disturbance of the cutaneous sensibility. All the symptoms gradually disappeared and she left the hospital; but nine days later she returned, because the swelling was as hard as ever. At the same time she complained of severe frontal headache, more especially on the right side. She complained also of the right cheek having a benumbed feeling, just like ice, touching it with the finger, however, making it feel as if burning, and there was also tingling of the part. Sensation was normal except in the sub-orbital region, where it was diminished. There was paralysis of the fifth nerve, pricking with a needle being neither felt on the right side of the face, nor on the right border of the tongue; taste being greatly diminished on the anterior half of the right side of that organ; and irritation of the nostrils not causing sneezing. There was also insensibility of the cornea and the sclerotic coat of the right eye. Sight was affected, for if the patient shut the left eye, she complained of seeing everything with the open eye as through a mist, but there was neither diplopia nor strabismus. As the pain disappeared from the right calf, the part was attacked by profuse sweating at night. The woman now developed head symptoms such as violent headache, vertigo, loss of sleep, nightmare, dilatation of both pupils, weakening of memory, hypochondria, and melancholia. Erythematous patches developed on the zygomatic processes, which disappeared, only to re-appear, however, next day over the cheek bones. Vision was very feeble, and deafness, particularly on the left side, became more marked. This deafness increased, while the sensory disturbances of the face gradually improved.

[1] Lancereaux: In Simon's *Thèse de Paris*, 1882, p. 68.

Skowronsky[1] has described the case of a patient who was seen twenty hours after poisoning by CO, and in whom there was swelling of the whole of the right side of the body, with paralysis of the right arm and of both legs. The œdema and paralysis quickly disappeared from all parts except the right arm, where it remained for a considerable time. Sensibility was unaffected, but the reflexes had disappeared, and the reaction of degeneration was present in all the muscles. The swelling was like that seen in elephantiasis. The patient, after a long illness, made a good recovery.

Sloughs and abscesses may develop. Bed sores are common, and generally appear over the sacrum, sometimes over the trochanters. Hasse[2] records the following facts regarding five soldiers who were exposed to CO. Two were found dead, a third died in convulsions, but the other two recovered, the one temporarily, the other completely. One remained unconscious for eight days and paralysed up to the twelfth day. On the sixth day the whole surface of the body became covered with pemphigoid bullæ which developed into ulcers. On the eighth day speech returned, although incompletely, but later on in the day the patient had an attack of convulsions. A fresh crop of bullæ developed, and a large slough appeared on the sacrum. The extremities were completely paralysed, as was also the bladder, the urine being ammoniacal and containing much sugar. The second man who survived regained consciousness twenty-four hours after the accident, remained very feeble, suffered from incomplete paralysis of the bladder, and, although he was only three days in bed a very large abscess appeared on the chest and on the left buttock.

Several cases are found recorded in which after the application of mustard plasters to the calves, a routine practice in CO poisoning at one time, there was considerable sloughing of the tissues underneath the areas of application, and the formation of a large ulcer which proved most intractable to treatment. Deep sloughs developed over both buttocks in a case described by Arnozan and Dallidet[3] which ended fatally, the *post-mortem* examination showing marked neuritis of the sciatic nerve. The reader is referred to the case reported by Alberti in which remarkable sloughing and laying bare of spinous processes in the neck and of bones of the skull from the occipital to the mastoid process is described (p. 241). In a case recorded by Marthen[4] of a

[1] Skowronsky: "Neuritis in Folge von Kohlendunstvergiftung." *Neurolog. Centralbl.*, 1901, p. 1071.

[2] Hasse: *Preuse. Med. Vereinszeitg. N. F.*, Bd. II., 1859, p. 35. Quoted by Leudet, *loc. cit.*, p. 520.

[3] Arnozan and Dallidet: *Journal de Méd. de Bordeaux*, 1883-1884, p. 30.

[4] Marthen: *Virchow's Archiv.*, 1894, Bd. 136.

woman of 31 who was found in a deeply comatose condition, small bluish-black stripes surrounded by areas of redness were found over the sacrum. After regaining consciousness, she complained of great pain in that region, the discoloured parts being particularly painful on pressure. Later, sloughs about the size of half-a-crown developed on the sacrum, and a month later a large abscess appeared on the left buttock. At the same time very severe diarrhœa, which lasted for a few days, set in, this being followed by an attack of pneumonia, from which the patient died three months after being gassed.

There may be numerous gangrenous spots, as in a case described by Jacob.[1] The patient, a woman, had marked trophic disturbances of the skin, with dryness and thickening of the epidermis, succeeded by profuse desquamation, swelling and stiffness of the knee, multiple areas of gangrene, eschars on the sacrum and on the trochanters, a feeling of profound weakness for months, and incessant and intolerable pains in the left thigh and leg.

In the case reported by Lancereaux[2] (p. 233) progressive weakness of the legs developed, followed by swelling, more especially of the right leg. As the œdema disappeared, an ulcer of the size of a crown appeared on the outer surface of the right leg at the junction of the lower third with the upper third. Laveran[3] reports two cases in which subcutaneous emphysema followed CO poisoning. In the first case there was right hemiplegia with hemi-anæsthesia, narrowing of the visual field, exaggeration of the reflexes, and the development of emphysema of the upper part of the chest and of the base of the neck. In the other case, where a soldier attempted suicide, the face was red and swollen, there was no paralysis in the limbs, and no anæsthesia, but fine crepitations of subcutaneous emphysema were found in the front region of the trunk up to the neck and in the back. When he recovered consciousness, the man was unable to give any description of his attempt at suicide, and failed to remember the name and address of the hotel in which he was found. He complained that a whitlow on his finger had not been dressed for eight days, whereas it had been dressed the day before. Later, he seemed to be able to read a book, but when asked the title he was unable to recall it. In a few days the emphysema gradually disappeared, and in a month the general condition vastly improved, but there was still loss of memory, and he was only able to reply in monosyllables. Another case of subcutaneous emphysema was reported by Duponchel.[4]

Glossy skin, which is so frequently found in nerve lesions since Paget

[1] Jacob. Quoted by Vialettes, *Thèse de Paris*, 1895.
[2] Lancereaux. Quoted by Simon, *Thèse de Paris*, 1882, p. 66.
[3] Laveran: *Bulletin de la Soc. des Hôpitaux*, July, 1890.
[4] Duponchel: *Gaz. Hebd.*, 1891, p. 8.

first called attention to it, has been reported by several writers as following CO poisoning.

Benson[1] reports a case where a crop of boils developed three or four weeks after the patient had been very seriously poisoned by gas, and others have reported cases of carbuncles. Besides, instances in which acne and purpuric spots have developed are not uncommon.

Tendon Reflexes.

Most authors have paid very little attention to tendon reflexes, and where their condition has been noted, the results have been by no means uniform, as in some cases they were found to be lost, in others diminished, while in a certain number they were unaffected. Avramoff,[2] for example, noted in three serious cases that they were preserved. Munzer and Palma,[3] Rendu,[4] Laveran,[5] and others have described cases where the reflexes were increased, and Brissaud[6] also found in a few instances that they were exaggerated, although in most cases they were lost. Usually in serious cases the cutaneous reflexes are preserved, while the deep reflexes diminish gradually till they disappear. It must not be forgotten, however, that in CO paralysis, unlike other toxic paralyses, the reflexes may be exaggerated.

Electrical Reactions.

As with the tendon reflexes very few authors have paid much attention to the electrical reactions. Where they have been noted we find very divergent results, in some cases contractility being recorded as abolished altogether, while in others it is stated as being preserved. What generally happens where paralysis has been present any length of time is, that the muscles lose their faradic contractility, or at least it becomes much diminished, while the galvanic current produces a slow worm-like contraction, with the anodal contraction equal to or greater than the kathodal. In very severe cases galvanic irritability may also be abolished. In multiple neuritis caused by various toxins, a loss of faradic and a considerable decrease in the galvanic excitability are generally found. Rendu pointed out in a case of paralysis of the extensors that the electrical reactions were identical with those found in lead poisoning, but in this case the continuous current was not used. Contraction to electricity reappeared before voluntary contraction.

[1] Benson: *Brit. Med. Journal*, 5th July, 1873.
[2] Avramoff: *Thèse de Nancy*, 1900, p. 54
[3] Munzer and Palma: *Zeitschr. f. Heilk*, 1894, p. 194.
[4] Rendu: *Société Médicale des Hôpitaux*, 13th Jan., 1882.
[5] Laveran: *Bulletin de la Soc. des Hôpitaux*, July, 1890.
[6] Brissaud: *Des Paralysies Toxiques*, Paris, 1886, p. 60.

Several authors have noted the reaction of degeneration. For example, in Boulloche's[1] case of paralysis of the deltoid muscle, faradic excitability of the deltoid was very much diminished (p. 244). The reactions in that case were as follows:—

Galvanic.	Anterior Deltoid.			
Sound side	K.C.C.	>	A.C.C.	2 m. ampères.
Paralysed side	K.C.C.	>	A.C.C.	6 ,,
	Posterior Deltoid.			
Sound side	K.C.C.	>	A.C.C.	4 ,,
Paralysed side	A.C.C.	>	K.C.C.	4 ,,

There is here modification of the galvanic contractility in quality and quantity as in the reaction of degeneration. Boulloche mentions that this reaction was absent in a patient who had hemiplegia following CO poisoning.

The following are the electrical reactions found in the case of hemiplegia of peripheral origin reported by Lereboullet and Allard[2]. In the right arm there was abolition of faradic excitability of nerves and muscles, with the exception of the posterior fibres of the deltoid and the pectoralis major. There was also diminished galvanic excitability, with slow vermicular contraction of the muscles of the arm, and an alteration in the normal polar reactions, the A.C.C. being even more readily produced than the K.C.C. The faradic irritability of the muscles of the leg and foot was very much diminished, but not entirely abolished. The polar reactions were almost equal in the muscles of the antero-external region of the limb; these muscles also showing vermicular contraction. There was a tendency to partial reaction of degeneration in the region of the external popliteal nerve. The farado-cutaneous sensibility, although diminished, was not abolished in the upper arm. In the lower limb hyper-excitability with the faradic current was noted. From their examination, the authors concluded that there was a neuritis involving both motor and sensory fibres of the terminal branches of the brachial plexus, and a neuritis of the motor fibres of the sciatic nerve, especially of the external popliteal branch. Rendu called attention to the fact that the peroneal muscles are the last to recover their power of contractility. Since they are often the first to be affected, Brissaud considered these muscles the most susceptible to CO poisoning.

Mental Disorders.

In many of the cases already described there have been prominent mental disturbances. Some of the cases were ushered in by shades of mental disturbance varying from marked restlessness, or wild excitement,

[1] Boulloche: *Archives de Neurol*, 1890, p. 219.
[2] Lereboullet and Allard: *Revue Neurol.*, 1899.

up to delirium and mania. One mental disorder, almost characteristic of CO poisoning as it is also of alcoholic neuritis, and which was so minutely described by Wilks, is the loss of memory relative more especially to time. All appreciation of time and place is lost; and paramnesia may also be met with, the patient describing in a graphic and detailed manner places he imagines he has visited, and people with whom he imagines he has just conversed. These symptoms have been described by Korsakoff as the "psychose polynévritique," and are generally spoken of in this country as Korsakoff's syndrome. To these mental disorders Dosseur[1] maintains we ought to add dementia, since a large number of cases of this have now been recorded.

Prognosis in CO Paralysis.

Attention has already been drawn to the marked fluctuations in the progress of all the symptoms in CO poisoning, the same being also found in connection with neuritis. The case of Bourru (p. 248) shows well the manner in which relapses may supervene when everything points to a rapid recovery. The bulk of cases probably end in recovery. It is impossible to state definitely which class of cases recover most rapidly, as there are the greatest possible variations in the duration of the affection even where the same groups of muscles have been paralysed. There is only a small number of recorded cases in which permanent lesions, as contractures, have resulted, or in which death has ensued. In some of the cases where the symptoms are grave from the beginning, as in some of the hemiplegias caused by such grave cerebral lesions as large hæmorrhages, the prognosis is very serious. Instances where there has been generalised and progressive paralysis, as in those recorded by Leudet and Lancereaux, generally end in death. Often in such cases dementia, or other grave mental disorder, has supervened some time before the end. Recovery may be long delayed in the more severe cases. These frequently become progressively worse for the first few weeks, then there is slight improvement, and then the condition remains stationary or there may even be a relapse. Once improvement has advanced to a certain stage, however, the progress to complete recovery may be very rapid. A few cases have been reported where the patient suddenly recovered, but in the great majority recovery is very slow.

Differential Diagnosis.

In the great majority of the cases where paralysis, nervous disorders, and disturbances of other organs are said to have followed poisoning by CO, it will not be difficult to differentiate these from those caused by other poisons. For example, it will be found an easy matter to put out of court nerve lesions caused by diphtheria, malaria, and other fevers,

[1] Le Dosseur: *Thèse de Paris,* 1901, p. 52.

rheumatism, gout, syphilis, etc.; and also such poisons as bisulphide of carbon, arsenic, mercury, and lead. In the paralysis of the musculo-spiral nerve it is bilateral in lead poisoning, and the supinator longus is unaffected; in alcoholic poisoning it is generally bilateral; while in CO poisoning on the other hand the paralyses are rarely symmetrically placed.

Diagnosis, however, is not always so easy as it appears at first sight. It must always be remembered, for example, that in a number of cases of paralysis, etc., following acute CO poisoning, the disease comes on a considerable time after the acute symptoms have passed off. This interval, during which the patient may appear to have fully recovered, may extend over a few weeks but generally lasts only a few days. Diagnosis will also be difficult in these cases where the nervous and other disorders have followed repeated exposure to small doses of CO, and where at the same time other agents, as alcohol, may also play a part. In certain occupations, too, other elements such as defective hygiene, etc., also intervene to obscure the picture, and the difficulty is always increased in such cases by the fact that opportunity for examining the patients only presents itself when the patient is in the cachectic state. In some of these cases, indeed, the patient was thought to be suffering from organic disease of the stomach, or from advanced anæmia.

Where the condition appears among young girls, CO poisoning must not be confused with chlorosis. In the latter, a disease of puberty, the illness does not last for prolonged periods generally, and is very amenable to proper treatment, whereas in CO poisoning it is very slow, gets progressively worse as long as the patient is exposed to the noxious influence, and is generally complicated by serious nervous disturbances which are not found in chlorosis. In all cases the occupation of the patient, and also the premonitory symptoms, will be a guide to the diagnosis.

With the enormous increase in the use of gas in industries, compensation cases are bound to appear in Court from time to time, so that it is very important in cases of paralysis, etc., to be able to affirm definitely whether such were caused by CO or not. To increase the difficulty of diagnosis, patients may put down their condition to some other cause, and thus the doctor may not be informed that their occupation exposes them to CO poisoning. For example, Simpson saw a patient, an ironer, who suffered constantly from severe headache, vertigo, visual hallucinations, etc., which symptoms were always worse during glossing and ironing, and were ascribed to the heat of the laundry. Simpson believed that the warm and close atmosphere in such cases is generally looked upon as the cause of all the troubles in laundry women. Certainly the conditions under which many of the patients work, some of which lead

the worker to indulge too freely in alcohol (and, as we have noted before, alcohol makes the patient more susceptible to CO), must not be lost sight of.

Are there any characteristic signs of the paralyses following CO poisoning which will assist in clearing up the diagnosis? The following features should guide us. The paralyses, with rare exceptions, are peripheral in origin, they are distinctly localised, are rarely symmetrical, and as Simon wrote in 1883, are distributed in a haphazard manner without following any rule, and without apparent reason. Another important feature is that, in some cases, the disorder jumps from one place to another, changing its position in a few hours. Simon, Vialettes, and others hold that the distinguishing pathognomonic sign of poisoning by CO is that the nerve is not attacked in all its functions, that is to say, there may be trophic and vaso-motor disorders without any interference with sensation and movement, and that the latter may also occur alone. This is only seen after CO poisoning, for in alcoholic poisoning trophic disturbances are always accompanied by motor and sensory disturbances.

The greatest difficulty in diagnosis will be found in distinguishing the paralyses brought about by CO and those caused by alcohol; and, it must be added, there is no well-marked boundary line between the two. Here the history of the case will be a guide, more especially if there are present characteristic dyspeptic symptoms, in particular, symptoms such as icterus, ascites, etc., denoting the presence of cirrhosis of the liver. The alcoholic patient also puts on flesh, whereas a marked feature in most CO poisoning cases is the loss of weight. In alcoholic poisoning tremors and cramps are frequent, the former attacking first of all the fingers and hands, then the feet, lips, and tongue. Disorders of speech may be found in both, but the convulsive attacks and the characteristic cerebral disorders (delirium tremens) found in alcoholism will be a considerable aid in clearing up the diagnosis. The paralysis produced by alcohol is generally limited to the extremities, and is usually accompanied by disturbances of sensation and vaso-motor disorders, whereas the prominent feature of CO poisoning is that the motor, sensory, and vaso-motor disorders may each appear by themselves, unaccompanied by the others. In alcoholic poisoning the trophic disorders are symmetrical, are localised to the extremities from which they progress upwards, and also appear in any position, perhaps far from the paralysed part. Œdema in alcoholism, for example, is always found in the extremities, but in CO poisoning it may be found in another region altogether. The cerebral disorders (the delusions, delirium, hallucinations of sight, etc., impairment of memory, up to actual dementia), are in a manner similar, but in CO poisoning these generally come on much more suddenly; and

there may be found, also, after the immediate symptoms of acute poisoning have subsided, an interval of a few days during which the patient appears to have completely recovered. We have already described the steppage-gait found in certain conditions following acute and chronic poisoning by CO, which is sometimes described as pseudo-tabetic gait, but in alcoholism it is quite different from this. It is much more clumsy, and is due to the foot-drop which is rarely found in tabes. In both there may be lightning pains, but the patient can generally stand quite steady with the eyes closed, and the pupillary signs are also absent in CO neuritis.

Disturbances of Sensory Organs caused by CO Poisoning.

Auditory Disturbances:—It has already been noted that a common complaint of patients exposed to CO gas is of noises in the ears, such as whistling, ringing, or tinkling. These sounds are soon replaced by a continuous booming or buzzing sound. These disturbances may last for a considerable time after recovery. Dr Motet,[1] when he found himself being overcome by CO, felt as if he had received two terrific blows on the ears, which were followed immediately by great tinkling and a feeling of intense vibration in the auditory apparatus. This tinkling was so loud and insistent as to keep him from sleeping for several nights. Where vertigo is a prominent after-symptom, it is often found that the patient complains at the same time of continuous pains in the ears. One patient informed us that he felt as if he had "a brass band in his head." Faure,[2] who paid particular attention to this symptom, made a minute and interesting analysis of cases which came under his own notice as well as of those he gathered from the experience of others. "First of all," he says, "confused beatings in the ears, which last for some time, torment the patient." At a certain stage these are exaggerated, and a dull and continuous vibration results which he likened to that of a carriage rumbling at a distance. This is complicated by beatings which are at first feeble and distant, but gradually approach nearer and become louder. "In the midst, then, of quietness, the patient is deafened by an incessant booming, which comes and goes, of reiterated piercing blows which in their turn become less and completely die away, at which point the patient remembers nothing more." The same phenomena occur after the patient regains consciousness, and may last for a number of days.

As has been noted, these auditory disturbances are frequently accompanied by vertigo and disorders in the equilibrium, staggering gait,

[1] Motet: Loc. cit., p. 259.

[2] Faure: *Archives Générales*, 1856, p. 37.

etc., and are probably caused by increase of tension in the semicircular canals.

Deafness.—In some cases there may be temporarily complete deafness which, however, passes off very quickly, but in some cases the deafness may persist for a long time. In the following case of *Bourdon*,[1] the patient after CO poisoning lost sight, speech, and hearing. He was unconscious for twelve hours; gradually movement and sensibility returned, but for four or five days he remained deprived of sight, speech, and hearing. Violent headache, giddiness, and booming and tinkling sounds in the ears were marked features. Two or three days later, when the symptoms had greatly lessened, the patient, after a fatiguing walk in the sun, was again more severely attacked by the same symptoms. Intelligence remained good. Five days after this relapse, he began to hear a little in the left ear, and on the following day also in the right. Twenty-eight days after the accident he was discharged from the hospital as cured. In another case recorded by the same observer, in which there were paralysis of the left arm and loss of speech, deafness was a marked symptom.

Kayser[2] published the following case. A woman, 36 years of age, along with her daughter, 11 years old, was poisoned by CO. The daughter died, but the mother survived although she only recovered consciousness after thirty-six hours. It was then found that she was quite deaf. She suffered also from headache and violent buzzing in the ears. For a short time after her return to consciousness, she showed maniacal excitement, accompanied by hallucinations of sight and hearing. She thought she heard the ringing of bells, the crying of geese, etc. There was also slight reduction of the power of vision and of sensation, and a feeling of numbness and heaviness in the legs. Four weeks later there was still difficulty in hearing, with buzzing in the ears. At the end of eight months she had made great improvement; the subjective sounds had disappeared, and her hearing was very much better. This woman up to the time of her accident had had perfect hearing. *Lancereaux* has also reported a case of chronic poisoning where deafness was a prominent symptom.

Disturbances of Vision.

During the time a person is being overcome by gas, he may find that his eyes become slightly painful, with smarting and lachrymation that the sight begins to fail, that motes float about in front of his eyes, or that sparks or flashes of light appear before them, and that everything then

[1] Bourdon: *Thèse de Paris*, 1843.
[2] Kayser: *Wiener Medizinische Wochenschrift*, 7th Oct. 1893, p. 1666.

begins "to go black." On recovering consciousness a peculiar rolling of the eyes is frequently seen, which may go on for a considerable time. Cases have often been reported where the patient, after recovering, complained that when he looked out of the window at objects, they appeared as if seen through a cloud or mist. In some cases there is a remarkable staring and startled appearance; in others the eyes are very restless, and there is constant blinking.

We have already drawn attention to remarkable *oscillations of the eye-balls* which have been described in several cases of poisoning by after-damp, in which the movements were incessant and continued for a few days. This phenomenon has been noticed in a most exaggerated degree in cases of producer and illuminant-gas poisoning. Such a case is described by Taylor[1], in which both eyes moved the whole meridian from right to left and from left to right, and never stopped for a moment. Nothing seemed to have any effect on this constant movement; "neither light nor darkness, irritation or repose, nor the internal conditions which governed the varying sensibility of the pupils, seemed in any way to modify the constancy of the motion, or relieve the monotony of its rhythm." In a case reported by Voss[2], the eyes moved from right to left like a pendulum, and when this motion ceased the movement started from above downwards. In the case of Barthélemy and Magnan, which we report in full (p. 301), the eyeballs for two days were agitated with convulsive movements either in a vertical or rotatory direction, these movements being so pronounced as to be perceptible under the closed lids. Bloch and others have also reported exaggerated cases of this which lasted for a number of days.

The *condition of the pupils* varies very much in CO poisoning. Occasionally in the comatose condition these are found to be widely dilated and insensible to light. In other cases they are irregular, but in the large majority of cases they are slightly dilated, and are found to react to light even when the patient is unconscious. Jaksch[3] says that in chronic poisoning the reaction to light is very sluggish. Morton[4] found in his cases that the pupils were dilated only in the most severe cases. In a few other cases, also very serious, there was considerable variation, the pupils being sometimes markedly contracted and at other times dilated. Of the seven patients admitted to hospital after the

[1] Taylor: "Poisoning by the Inhalation of Coal-Gas." *Edin. Med. Jour.*, 1874, Vol. XX., p. 22.

[2] Voss; "Ueber Tetanie bei Kohlendunstvergiftung," *Deutsche Med. Wochenschrift.* 6th Oct. 1892, p. 894.

[3] Jaksch: *Die Vergiftungen*, 1901, p. 257.

[4] Morton: "Notes on Cases of Coal-Gas Poisoning," *St. Barth. Hospital Reports*, 1885, p. 73.

Crarae disaster, it was found that in every case the pupils were dilated but responded to light, and that in the case which ended fatally, the pupils contracted before death. A few cases have been recorded where the pupils were contracted to a pin-point size, the case resembling one of opium poisoning or bleeding into the pons Varolii. These cases very often end fatally. Bloch[1] reported a case where the pupils were very small, and where there were continual spasms of the muscles of the upper part of the body; the patient dying on the second day. Posselt[2] also reported a similar case. In a case of double facial paralysis following CO poisoning reported in the Factory Reports, in which there also existed inability to walk, intense formication in the arms and legs, cyanosis, and great sleeplessness, the pupils were markedly contracted and remained so for a few days. Golding Bird[3] describes a most interesting case where the pupils were alternately contracted and dilated at intervals, till death occurred two days later. J. H. Benson[4] also reports a case where there was alternate pupillary contraction and dilation. This patient recovered. Marthen[5] reported a peculiar case where, in a man of 42 after CO poisoning, the right pupil, which was much smaller than the left and not completely circular, did not react to light.

Exophthalmos and paralysis of muscles of eye following CO poisoning.—Cases of exophthalmos have been described by a few writers, as Friedberg and Pokrowsky. It was also present in the following remarkable case of paralysis of the eye-ball following CO poisoning reported by Knapp[6]. A man, 27 years of age, had been repairing his hot-oven, but had overlooked a small fissure, and the fumes escaping into the room in which he was sleeping, rendered him unconscious. He remained unconscious for two days, and for ten days was so ill that his life was despaired of. When he recovered consciousness, it was found that the external and internal muscles of both eyes were completely paralysed, the power of accommodation for near vision being also lost, and there were considerable photophobia and a certain amount of paralytic exophthalmos. Graef's sign was present; that is, on lowering the eye-balls the eye-lids do not follow as in the normal conditions. During the first week he could not look upwards at all. Ophthalmoscopic examination showed nothing abnormal, and no signs of paralysis or of paresis appeared in any other part of the body. The paralysis proved most refractory to treatment, and although after two months the ciliary muscles and sphincters of the

[1] Bloch: *Loc .cit.*, p. 16.
[2] Posselt: *Wiener. klin.*, *Wochen*, 1893, Vol. VI., pp. 376, 399.
[3] Golding Bird: *Guy's Hospital Reports*, Vol. IV., p. 84.
[4] J. H. Benson: *British Medical Journal*, 1873, Vol. II., p. 24.
[5] Marthen: *Virchow's Archiv.*, Vol. 136, p. 536.
[6] Knapp: *Archiv. für Augenheilkunde*, 15th Jan., 1880, p. 229.

pupils almost regained their normal condition, considerable weakness of the superior, external, and inferior recti persisted, while the internal recti remained completely paralysed. The result was that there was very marked double outward squint. Tenotomy was afterwards performed, but improvement was very slow.

Blindness following CO poisoning—.After exposure to CO, a patient on regaining consciousness may complain of complete loss of sight. This condition may last for a short time after that, but it generally disappears. Hilbert, [1] for example, reported a case where after the patient had been deeply comatose he developed xanthopsia (yellow vision), accompanied by diminution of the visual acuteness with narrowing of the visual field, which lasted little more than a day; and Witter of Dublin, who was one of the first to experiment with CO and was himself rendered unconscious, after being brought round was affected with total blindness which lasted some time. We have already alluded to the case reported by Bourdon in 1842, where the patient lost his speech, sight, and hearing (p. 261).

We have made a careful scrutiny of the literature of CO poisoning, but have been able to collect only a small number of cases where the ocular lesions were serious. Of these, probably the following are the most interesting. One of the earliest cases reported was by Illing,[2] in 1873, of unilateral hemiopia which followed poisoning by charcoal vapour. When the patient recovered consciousness, it was discovered that he was completely blind. As his general condition improved, vision also improved considerably, but everything appeared as seen through a mist. This disappeared in three days, but considerable diminution of visual acuteness still remained. Ophthalmoscopic examination revealed an annular staphyloma of the left eye, with a yellowish slightly-pigmented spot situated near the border of the yellow spot. The visual acuteness was normal in the right eye. Comby [3] describes the case of a man, 23 years of age, who several days after having been poisoned by CO and on regaining consciousness, was found to be completely blind. He could not move his limbs in bed, and there was incontinence of urine and fæces. For the first eight days he could not see anything, no matter at what distance the object was placed, but at the end of that time his sight gradually came back, although it remained disturbed for a long time. Movements in the limbs of the left side returned, but paralysis persisted on the right side, the face being unaffected. Fifteen days afterwards he began to move about but dragged the right leg, while he

[1] Hilbert: *Jahresb. Wirch*, I., 1896.

[2] Illing: *Allgem. Wien. Med. Ziet*, 1873, 23-25, quoted by Abramovitsch, *Thèse de Lyon*, 1898.

[3] J. Combv: *France Méd.*, 1882.

could not use the right arm. He still complained of motes before the eyes, but gradually improved thereafter, and left the hospital completely cured.

Arnozan [1] reports a case of a man, 46 years of age, in whom the poisoning was not nearly so serious, and who was quickly brought round, but who suffered for a considerable time from general muscular paresis, and from disturbance of vision which remained permanently. For several hours after poisoning the patient was completely blind, but vision came back little by little, this improvement ceasing, however, at the end of eight days. Twenty days after the accident his vision was found very imperfect, and he saw objects as if through a haze. With the ophthalmoscope, on both sides, but more especially on the left, optic neuritis was found, and the beginning of papillary atrophy. Under treatment his sight continued to improve up to a certain point, but after three months the above-mentioned conditions still persisted, if anything even more exaggerated in the left eye.

Audry [2] also reported a case where well-marked optic atrophy appeared in a woman who was poisoned by CO. In this case there were violent headache, vertigo, general weakness, remarkable trophic disturbances (virulent inflammation of the gums, ulceration of the tonsils, reddish patches on the forearms with subcutaneous induration, some of which afterwards sloughed, the sloughs being surrounded by a zone of anæsthesia), retention of urine, and mental disturbances of a hysterical nature.

The following case recorded by Purtscher,[3] in which there was permanent disturbance of vision, is interesting not only from its variety of symptoms following CO poisoning, but from the fact that it closely resembled recorded cases of toxic poisoning from such poisons as carbon bisulphide, alcohol, nitro-benzene, etc. A clergyman, 60 years of age, was accidentally poisoned by gas in a hotel. He was unconscious for one day, and for ten days quite blind. Then very slowly, and only to a very slight extent, his vision returned. For weeks he was mentally deficient, and six weeks after his accident his mental power, and especially his memory, more particularly for names and places, was still very weak. He was awkward and confused and quite indifferent to his surroundings. His speech and, indeed, all his movements were slow. His genera health was good. The pupils were slightly dilated, but there was no strabismus. Acuteness of vision in both eyes was $\frac{3}{12}$, and was not improved by spectacles. Nothing abnormal was found on ophthalmo-

[1] Arnozan : *Jour. de Méd. de Bordeaux*, 1883.

[2] Audry : *Lyon Médical*, 21st March, 1897.

[3] Purtscher: "Ueber die Einwirkung von Leuchtgasvergiftung auf das Sehorgan," *Centralblatt für praktische Augenheilkunde*, Aug. 1900, p. 225.

scopic examination, except that the arteries were a little more tortuous and contracted than normal. His colour sense was normal. There was marked narrowing of the visual field. This was preserved in front and above, but below and to the sides it was lost. Two months later his mental condition was still very weak, but his memory and speech had slightly improved. His visual acuteness had improved very much. The pupils were still a little dilated and reacted slowly to light. With the perimeter it was found that the left half of the field was still wanting, and also the lower half of the right. Ophthalmoscopic appearances were practically normal. Reflex sensibility to light was lost in the affected parts. Two or three months later the visual field was still found narrowed, although acuteness of vision was now almost normal. In this case, then, following CO poisoning there was a double-sided amaurosis, which gradually cleared up, leaving homonymous hemianopia (lateral in both, complete in the left, and incomplete in the right), with normal acuteness of vision and preservation of the colour sense, accompanied by mental disorders such as weakening of the intelligence and of the memory for places. Purtscher believed that the visual disturbances in this case were caused by small hæmorrhages or areas of softening in the visual centres or even in the cortex.

A very similar case was reported by Lochte[1] in 1905. A man after being seriously poisoned by CO had difficulty in finding his way about, requiring assistance when he walked about the streets. Looking at the sign board of an electric car he was only able to distinguish a word here and there. Examination showed nearly normal acuteness of vision, but the field of vision was considerably contracted. Nothing abnormal was disclosed by the ophthalmoscope, and there was no other ocular disturbance. The diagnosis was double-sided homonymous hemianopia, and as the field of vision gradually extended on both sides, hysteria must be put out of court in diagnosis. Lochte regarded the restriction of the field of vision as due to a hæmorrhage into the cortex of both occipital lobes. Schmitz[2] also reported a case where after CO poisoning the field of vision was much contracted, and the acuteness of vision was diminished to $\frac{1}{6}$.

In all these cases there was no disturbance of the colour sense. But in the following cases, the diminished sense of colour and especially the greatly diminished faculty of recognising green, make the analogy of the ocular disturbances resulting from CO poisoning and of those after

[1] Lochte: "Fall von Sensorischer Aphasie und doppelseitiger homonymer Hemianopsie nach Kohlendunstvergiftung," *Muench. Med. Wochenschr*, 1905, p. 2204.

[2] Schmitz: "Ueber die Einwirkung der Kohlenoxydgas-vergiftung auf das Auge," *Festschr. Wiesb. Bergmann*, 1893.

poisoning by alcohol, carbon bisulphide, and other toxic substances appear very striking. In the first, a case of Schmitz, immediately after the accident the pupils reacted very sluggishly. There was well-advanced concentric diminution of the field of vision (to 5 degrees), with a disturbance of the sense of colour. For the first two weeks the large retinal veins were distended, the arteries slightly contracted, and the optic discs had a greyish-white, opalescent appearance. After four months the acuteness of vision was normal, and on examination after a lapse of a few years the optic discs appeared normal, and there was no loss of colour.

Probably one of the most outstanding cases in literature, showing not only the characteristic nature of the multiple neuritis which follows CO poisoning, but also the widespread distribution of the lesions in many cases, its want of conformity to any type, its asymmetrical character, and certain ocular disturbances, is recorded in the following facts by Schwabe.[1] A man, 36 years of age, was poisoned by coke-oven gas. He was brought home in a delirious state which passed into coma, in which condition he remained for a week. There were neuralgic pains and considerable loss of power in both legs. This was more marked in the right leg which felt cold from the knee down, the foot being cyanotic and swollen. He also complained of pain, sensory disturbances, and loss of power in the right arm, the inferior branches of the left facial nerve being also affected. On examination of the eyes serious defects were discovered. Visual acuteness was much diminished, as also the sense of colour—this latter being much more marked in the left eye. The perception of green colour was most affected, that of blue being little disturbed. As in Knapp's case there was also paralysis of accommodation, the left eye being more affected than the right. A very important point in this case was the fact that the refraction of the eyes changed very considerably when they regained their function and the muscles their tonicity. They were at first markedly hypermetropic, but this greatly decreased in the course of weeks. Examination by the ophthalmoscope showed at first in both eyes a blurring of the borders of the optic discs, which were swollen. In the right there was, besides, a general redness of the disc, which was also seen in the left in the upper inner square, but the inflammation disappeared later and the borders of the disc became sharply defined.

A case, especially interesting from the pathological conditions found at the *post-mortem* examination, is described by Sibelius[2]. A lad of 20 was

[1] Schwabe: "Ein Fall von multipler Neuritis nach Kohlenoxydvergiftung mit Betheiligung der Sehnerven," *Muench. Med. Woch.*, 1901, p. 1530.

[2] Sibelius: "Zur Kenntniss der Gehirnerkrankungen nach Kohlenoxydvergiftung," *Zeits. f. Klin. Med.* 1903, p. 3.

exposed for five minutes only to CO. He became unconscious, his pupils were dilated and did not react, the left seventh nerve was paralysed, and it was found that he was completely blind in both eyes. Weakness of the extremities soon developed, followed by epileptiform seizures and dementia. The ocular symptoms persisted till death, which took place three months after poisoning. The lesions found by Sibelius at the *post-mortem* examination explained the development of blindness, for the cortex was softened to a large extent in both occipital lobes, especially in the middle, almost completely involving the calcarine fissure.

Bouchereau and Raffegeau[1] had previously reported a similar case in a man of 67, in whom there was dementia which set in immediately after the poisoning. There was loss of sight, which began in the upper parts of the retina. Careful examination by Kalt demonstrated a considerable degree of interstitial neuritis of the optic nerves. Kalt came to the conclusion that there were diffused lesions in the occipital lobes. The fact of the lesions being central would also explain two of the other mental symptoms met with. In Croizet's[2] case, in which there were pronounced mental symptoms, photophobia was present, the eyes requiring to be shaded, as bright light considerably disturbed the patient, and the sight was much affected. The patient was quite unable to distinguish men and objects; these he described as "vague phantoms which I see moving about me." After a few months he could see very little. He had lost the perception of relief, no longer appreciated the distance of an object or its position, and often let fall to the ground articles which he believed he was placing on the table. In another similar case reported by Raffegeau, the blindness was considered to be cortical in origin, and in a case of Friedenwald,[3] where there was a double-sided hemianopia, this condition was looked upon as the result of lesions, either hæmorrhages or areas of softening, in the cortical sight centre.

A case of optic atrophy, following the inhalation of the fumes from blasting by nitro-glycerine, has recently been reported by Dr Reid Pirrie.[4] For ten months before he stopped work, a miner had found the fumes very irritating, causing headache, sneezing, and vomiting, and occasionally epistaxis. His previous health had been excellent. The report was as follows:—"Patient is quite blind. General diseases which might cause optic atrophy are excluded by examination; no constitutional disease; heart and kidneys apparently normal. On ophthalmoscopical examination double optic atrophy found. Both right and left discs are

[1] Raffegeau: *Annales Médico-Psych.*, May and June, 1889.

[2] Croizet: *Thèse de Paris*, 1903.

[3] Friedenwald: *Archiv. f. Ophthalmol*, 1908, Bd. XXIX., p. 224, quoted by Sibelius.

[4] R. Reid Pirrie: "Nitro-glycerine Poisoning," *The Practitioner*, 1912, p. 259.

absolutely white; and the vessels smaller than normal." Dr Haldane, to whom the case was referred, although he had not heard of any case of optic neuritis following CO poisoning, said "It certainly looks as if these cases are due to CO, although possibly some other associated gas or vapour may be responsible. Anyhow, they do occur among men exposed to the products of imperfect combustion." Dr Judson Bury, who also examined the patient, agreed that "the optic atrophy could have been caused by the fumes of nitro-glycerine, carbon monoxide, and the fumes produced by the imperfect combustion of the explosive."

We should add that in nearly all the cases we have come across in literature, where the ocular disturbances following CO poisoning were marked, serious mental disturbances such as delirium, confusion of mind, paralysis (hemiplegia, etc.), loss of memory, dementia, etc., retention or involuntary passage of urine, etc., were usually concomitant.

Speech.

When a patient has been unconscious for any length of time, it is sometimes found on his recovering and attempting to speak, that his speech is affected. It is blurred and indistinct, or the words are chopped short and run into each other in a peculiar manner. In one patient we attended, who also suffered for a considerable time from partial loss of memory and dulness of cerebration, this symptom was prominent; indeed, the most remarkable feature of the case was the curiously blurred and gulping character of the speech; and for two or three months after, one could hardly make out a word he said. On making enquiry among medical men and others who have had considerable experience of CO poisoning, we have found that the blurring of the speech and curious gulping method of articulation is not an uncommon sign. In most cases the patient, on recovering consciousness, is only able to speak very slowly, as if he had great difficulty in finding words, and he frequently stammers. Probably this is owing to the condition of the brain: the patient can only think very slowly; is slow to understand; and the same sentence has, perhaps, to be repeated several times before he is able to grasp its meaning. This, with the dull, heavy, confused look, and, perhaps, the blurred speech, as well as the vertigo and powerlessness of the limbs giving rise to the staggering gait, all go to complete a picture of an intoxicated person. As we have already pointed out, such cases have frequently been mistaken for persons who are just recovering from a drunken bout. Chevalier [1] describes a case of difficulty in speaking after CO poisoning. In this case the tongue appeared as if paralysed, and the patient complained that she felt as if her tongue were thicker and bigger than usual. In other cases, it has been noticed that the patient

[1] Chevalier: *Loc. cit.*, p. 75.

can only speak in a hesitating and imperfect manner. He leaves sentences unfinished, lops syllables off words, and has difficulty in articulating certain letters.

Attention has already been drawn to what must be regarded as comparatively common in CO poisoning, viz., *a tendency to repeat constantly certain words or even phrases.* In certain cases of prolonged fatigue, it is well established that this curious reiteration of words occurs; and Mosso has pointed out a similar condition in persons who had been performing hard work in rarefied air. This repetition of words and sentences was a marked feature in a case recorded by Trénel[1] of a woman of 21 who was in a comatose condition for six hours after poisoning by CO, and who, on recovering, developed a severe convulsion. When she came to herself she was able to give her proper address, but her answers otherwise were incoherent. A most remarkable mental condition then appeared. During the next few days she became very excited and restless, gesticulating frequently in a wild manner, making grimaces, and behaving in a quarrelsome fashion, as, for example, beating any child she could get hold of in the street. She constantly repeated the same sentence: "Carnot est mort, tué par un Italien." Every now and then she would count up to thirteen, sometimes many times in succession, and went over the calendar, also, with many repetitions.

Becker[2] holds that any disturbance of speech after CO poisoning seldom lasts very long. In his case of disseminated sclerosis following CO poisoning, speech was markedly affected and remained so for a considerable time. At first the patient was only able to babble like a baby, and although he improved slowly, even after a considerable time he was not able to pronounce certain words, more especially those of more than one syllable. In this case the speech was much more affected at the beginning of the illness than it was later. The act of speaking appeared difficult to him. He spoke in one tone, sometimes with a nasal quality, and very slowly. Afterwards he could pronounce polysyllabic words quite correctly but slowly. Often at a later stage the speech would become singing or intoned at the end of a sentence. In another case of disseminated sclerosis by Avramoff, the speech was found to be typically scanning. Some patients after gassing, as has been noted, become very talkative, speaking generally, however, in an incoherent manner. In the type of case just described, we find that speech generally returns to its normal condition, and that in a few months at the very most it is as good as ever. Sir Thos. Oliver,[3] however, has reported two cases where the defect persisted for more than two years. In these cases speech was

[1] Trénel: *Gaz. hebd. de Méd. et de Chirurg.*, 1895, 30, p. 351.
[2] Becker: *Deutsche Medicinische Wochenschrift*, 1889, p. 541.
[3] Thomas Oliver: *Diseases of Occupation*, p. 60.

affected much in the same way as in general paralysis. There were also present nystagmus, incomplete loss of power in limbs, with development of steppage-gait and certain well-marked mental symptoms.

Aphasia.

Sometimes true aphasia is found after CO poisoning, and in such cases it is found that recovery is much slower than in the previously-mentioned cases. In certain cases of hemiplegia described by various authors, as Laroche, Simon, Lereboullet and Allard, true aphasia has been met with, some of which cases have been described under paralysis. In a case described by Bourdon, where paralysis of the left arm was found in a strong young woman poisoned by charcoal fumes, the patient was speechless for a day after the accident, there being also pronounced deafness. We have already described another case by Bourdon where there was loss of vision, of hearing, and of speech (p. 261). On the sixth day after the accident, when the patient again began to speak, he almost exclusively used vowels. In about a fortnight, however, speech had returned to its normal condition. In this case during the aphasic stage the patient was able to understand perfectly all that was said to him, and was able to make himself understood by writing.

In many of the cases in which serious mental disorders follow CO poisoning, it is found that there is a longer or shorter interval of freedom from symptoms, a quiescent period in which the patient appears to have completely recovered from the effects of the poison. This interval was short in the following case of Knecht,[1] but lasted longer in our case of amnesic aphasia. On 15th February 1895, a man, 30 years of age, was brought to hospital in a condition of complete unconsciousness. He had always been a strong, healthy, sober man, whose only illness had been an attack of syphilis in 1889, from which he had completely recovered. He had been employed in a newly-built but poorly-ventilated factory, in which two to three coke-ovens had been burning for a few weeks. On the day of his accident he remained at work longer than the other men, and became sick. He succeeded in getting out into the yard, where he collapsed, and where he was discovered in an unconscious condition. Later, he regained consciousness, spoke in his usual way, and was able to walk to his home, which was near. The following day he was found unconscious, with the lower part of the right side of the face and the right arm and leg completely paralysed. In a few days he became conscious, when it was found that sensation was very much diminished in the paralysed parts of the body. Sensation, however, was quickly regained. The patient also suffered from motor aphasia. He was

[1] Knecht: "Zur Kenntniss der Erkrankungen des Nervensystems nach Kohlenoxydvergiftung," *Deutsche Med. Woch.* Aug. 1904, p. 1242.

could, however, understand spoken and written words. He was quite unable to repeat words spoken to him, or to read aloud what was written. He quickly recovered the power of the leg, but the paralysis of the arm and the aphasia remained, and after three months his condition was practically unchanged.

Cases of aphasia have been described in which, at the *post-mortem* examination, a gross lesion was found in the brain. Poelchen [1] reports such a case of a woman, who, after CO poisoning, was unconscious for two days, but who recovered so rapidly, with the exception of her speech which was much affected, that she was able to resume work in a week's time. Three weeks later certain mental symptoms developed; she became like a weak-minded child, her speech became slower and slower and more difficult till it stopped altogether, and some time later she died. Scott and others describe similar cases. In all these there was well-marked dementia and, generally, paralysis of some kind.

In the following two cases however, this was not so. In one which came under our notice the patient was a most intelligent engineer, who, on coming to consciousness after prolonged exposure to blast-furnace gas containing about 26 per cent. of CO, spoke in an incoherent manner, and was violently delirious. A few hours afterwards, however, he began to talk quite rationally. Suddenly on the third day his mind became a total blank. But in this condition there was still some capability to form words, for he could say words such as "yes" and "mother," therefore it was not a case of complete motor aphasia; but, owing to the condition of his brain, he had practically to learn speaking over again like a child; he had to begin with the alphabet and learn to read, as he had also to do with his trade. Here the loss of speech was due to the loss of memory (amnesic aphasia).

The only other similar recorded case is that described by Le Dosseur,[2] which presents so many interesting features that we give it in detail. A woman, 44 years of age, healthy and non-alcoholic in habit, became depressed and melancholic, and attempted suicide by inhaling the fumes from a charcoal stove. Five hours later she was found, and was brought round with some difficulty. She then gave a perfectly clear and detailed account of how she had attempted suicide, A most unusual form of paralysis was, however, found, viz.:—paralysis of the Duchenne-Erb type, the deltoid, brachialis anticus, biceps, and supinator longus muscles being involved, but there were no tremors or trophic or vaso-motor disturbances. Elbow reflex was abolished on the same side. She left the hospital in a few days, but came back four days later as she and her

[1] Poelchen: "Gehirnerweichung nach Vergiftung mit Kohlendunst," *Berlin. klin. Wochenschr.*, 1882, No. 26, p. 397.

[2] Le Dosseur: *Thèse de Paris*, 1901.

family were greatly disturbed by certain mental changes which had developed, such as loss of memory and disturbances of speech. She had formerly been able to speak very well, and had been able also to give a detailed account of all the events of the days preceding her attempt at suicide. Nine days after the poisoning by CO, she suddenly perceived on waking after a good and quiet night's rest, that she had difficulty in finding and speaking words. She was unable to form phrases, and although she made great efforts to remember the proper word she wished to use, she was unsuccessful, while constantly, also, she made mistakes in choosing her words. Now her memory showed itself at fault, for she no longer remembered events of the last few days, nor those of the two or three days preceding her attempt at suicide. Her mental condition after the accident was interesting. In contrast to the sad, melancholic, restless and emotional mental state in which she had previously been, she was now singularly apathetic, and it became impossible to arouse her interest or attention. She passed days sitting in a chair staring into vacancy. She did her ordinary duties in the hospital ward, dressed herself, took her food, found her way about, and recognised the members of her family, attendants, doctor, and others. But the events of the eight days succeeding her attempt at suicide had been erased from her mind. She had forgotten, for example, her first stay in the hospital, her departure, and her return on three separate occasions for electrical treatment.

The disturbance of speech was regarded by Le Dosseur as the amnesic form of aphasia. There was no dysarthria, as pronounciation of words was easy. She understood perfectly well what was said to her. The memory of words alone was disturbed. The image of the object was there, she recollected perfectly its character, but she was not able to pronounce its name, or making a mistake, she would use one word for another. She sometimes started sentences but was unable to complete them, being stopped by a word she could not remember, although, as Le Dosseur adds, one felt that she had a perfect idea of the word she was hunting for. If she were handed or shown an object, for example, she would give it the wrong name, but as soon as she pronounced the word she herself appeared to see that it was wrong, and although evidently making every effort, failed to remember it. She would then give way to anger and despair. On being then told the desired word, she at once recognised and repeated it, but soon again forgot it. Asked to repeat a very simple phrase, she misplaced the words after one or two repetitions and stopped. There was no word-deafness, as she heard and understood everything that was said to her. Her comprehension was, however, a little slow, and a certain interval of time elapsed before she answered. Examination of unable to speak; when he tried only a senseless babble resulted. He

the eyes showed nothing abnormal; there was no visual disturbance, but *word-blindness* was fairly marked. She could not pronounce some of the words she saw, and she did not understand the sense of the words or, at best, only very imperfectly. Sometimes she had a faint idea of what the written lines signified, but it was impossible for her to convey that idea. When asked to give the meaning, she invariably answered "I know, but I cannot say." *Agraphia* was also present; formerly she could write well, but since her accident writing became impossible. She remained in the hospital for six months without her speech defects being remedied in any sensible degree.

Knecht [1] has published in detail the following case in which disturbances of speech were present, and in which many unusual and interesting points may be noted. On 18th November 1903, a woman, 56 years of age, and her husband were poisoned by CO. When discovered, the man was dead, and the woman had been in an unconscious condition for twenty-six hours. After three hours she became conscious, but was quite benumbed and spoke with difficulty, only being able to utter a few short words. The whole face was cyanotic and swollen on the right side. Both hands were also swollen and cyanotic, and bluish-coloured swellings were found over the right malar bone, right knee, and the upper part of the sternum. The urine was drawn off, and was found to contain albumen but no sugar. The following day her pulse was 28, soft and weak. There were no sensory disturbances except in the hands, and movements were normal, as were also the patellar reflexes. On the third day the whole of the right leg was swollen, and the lower part of the arms became cyanotic and œdematous, while blisters of the size of pigeons' eggs developed over the lower parts of the arms and hands, and on the right knee. On the forearm these were distributed in parallel rows, following the lines of distribution of the ulnar and radial nerves. Two days later, areas of redness with blisters developed on the right shoulder, and the whole of the right arm was much swollen but painless. From the beginning there had been paræsthesia in the hands, a feeling of irritation and itching in the left hand and prickling sensations in the right. For several weeks the stereognostic sense was wanting, and owing to this she conld do little with her right hand. The œdema entirely disappeared in the course of ten days. Although she was now much more rational, her memory was still poor, and she had great difficulty in speaking, answering questions only in the briefest possible way. On the 22nd January she became very restless, and that night slept very badly. In the morning when the doctor questioned her, she understood perfectly at first what was said and replied correctly, although in monosyllables. Then suddenly her answers became indistinct and incoherent, and she

[1] Knecht: *Deutsch. Med. Woch.*, 1904, p. 1242.

could only make out letters, with here and there a syllable. She could not tell the names of objects shown her, although later it was discovered that she had known the names but could not utter the words. Three quarters of a hour later she was able to speak again, but was still confused. After four hours she became quite rational, but further changes had now developed. There was hemiplegia of the right side of the body with complete paralysis of the right side of the face. In the arm sensibility for pain and temperature was lessened, but in the leg and all other parts of the body sensation was normal. Next night pneumonia in both lower lobes of the lungs developed, from which she made a good recovery in ten days. By the 26th January, sensation was normal in all parts except in the palm of the right hand, in a third of the back of the hand, and in the fingers, which were markedly contracted, extension of which caused her pain. This contraction disappeared later. In a fortnight her walk was normal, and she had regained the power of the right arm except the hand. By the end of December marked tremor had developed in both arms and hands, and also in the body whenever she became excited, as, for example, when she was being medically examined. There was no atrophy of the muscles. Her mental condition was now very much improved, the only defect being slight dulness at times. In this case, then, there was not only multiple neuritis, but symptoms caused by a central lesion, which must have been small, as the symptoms disappeared so quickly.

CHAPTER XI.

MENTAL DISORDERS FOLLOWING POISONING BY CARBON MONOXIDE.

It has already been pointed out that Bourdon, in 1843, was the first to attempt a systematic examination of the nervous and mental disturbances produced by CO poisoning, and several of his most interesting cases have been cited at some length. From time to time subsequently, reports have been made of cases in which mental symptoms were a prominent feature, but it is only comparatively recently that the peculiar action of CO on the intellectual faculties has been systematically studied; indeed, many modern medico-legal works ignore altogether this important aspect of gas poisoning.

In 1869, Moreau (de Tours) in his work *De la folie névropathique* (*vulgo-hystérique*), recognising how often mental disorders followed chronic poisoning, wrote as follows:—"Often in the course of our studies have we thought of treating *ex professo* the cerebral disorders due to the action of CO under the group Folie des Cuisiniers." In 1876, his son published a thesis entitled "Des troubles intellectuels dus à l'intoxication lente par le gaz oxyde de Carbone," in which he reported a considerable number of cases of chronic poisoning. In many of these cases the mental disorders were transient in character, passing off in a very short time, but in certain others, a difficulty is experienced in deciding whether or not alcohol played the more prominent rôle in the production of the mental disorders.

In 1882, Poelchen[1] contributed an important article on this subject. He collected all the known cases in literature, reviewing more particularly the work of Klebs and Simon, and added a few interesting cases of his own, in which areas of cerebral softening were found at the *post-mortem* examination, and which helped to explain many of the mental phenomena met with. Non-medical observers have long recognised that mental effects may and do follow CO poisoning, and Poelchen states that, in East Prussia, several persons had declared to him that people who had been seriously poisoned by CO, but whose lives had been saved, would become imbeciles. Three years after Poelchen's article appeared, Rouillard,[2] in his "Essai sur les Amnésies," reported another interesting case of CO poisoning in which a woman, who was markedly predisposed

[1] Poelchen: *Virchow's Archiv.*, Vol. CXII., p. 26.

[2] Rouillard: "Essai sur les Amnésies," Paris, 1887.

by her family history and nervous constitution to mental disturbances, developed maniacal excitement of a transient character with serious loss of memory.

In 1885, W. Chardin published a thesis on " Les Affections Nerveuses d'Origine Oxy-carbonique et sur les Alterations des Centres Nerveux dans l'Intoxication de l'Oxyde de Carbone," in which he collected together all the cases which had appeared in literature. In addition to these, we have already called attention to cases by Gnauck, Huchzenmeyer, Rochelt, Appolzer, and others. In 1885, Thomsen described a case of CO poisoning in which there were pronounced mental symptoms, the patient for some time suffering from acute delirium, which afterwards developed into a condition of dementia.

Cacarrié in 1887, in his " Essai sur les Amnésies Toxiques," pointed out that loss of memory, produced by CO gas, differed from that caused by lead and by other forms of poisoning in that it appeared suddenly and did not come on slowly and progressively. He described at length a most unusual case by Bouchet, where, following poisoning by CO in a girl who was an epileptic and who also suffered from melancholia, there was loss of memory of events stretching over a considerable period prior to her attempt at suicide. In the following year, Pouchet, Bouchereau, and Briand described mental disorders, more especially loss of memory, due to the same cause.

But it was left to Briand, in 1889, to make a thorough and detailed examination of the nature of the loss of memory and other mental phenomena following CO poisoning. He described two cases which had come under his own observation, and called attention more particularly to the retrograde characters of the amnesia, and to certain important medico-legal points which might result from this loss of memory. Later in the same year, he published another case in which loss of memory was the most prominent mental symptom. In the discussion which followed on Briand's paper, Beauvais, Bouchereau, and Brouardel mentioned cases which they had encountered. Musso, in the same year, published one of the very few papers which have appeared dealing with serious cerebral disorders following chronic CO poisoning. He described five cases in which marked psychical disturbances were met with. Two of the cases recovered, but the others became imbeciles, and before death occurred showed a typical picture of *dementia paralytica*. Charcot about the same time described the only case in literature where *astasia abasia* followed acute CO poisoning. As very few observers have paid any attention to the microscopical changes found in the spinal cord after CO poisoning, an article by Rokitansky which appeared at this time is interesting.

In 1891, Cramer published an article dealing with the pathological

changes in the brain, in which he raised very important points in the ætiology of CO poisoning, and in the same year, Schwerin contributed a study of the pathological lesions found in the brain and their significance. The following year, Koch published a dissertation "Zur Encephalomalacie nach Kohlenoxydvergiftung," in which he described at length the various pathological lesions found in the brain; Ruata described a case of acute delirium; and Fallot, one in which retrograde amnesia was the most prominent mental disorder. In 1893, important contributions appeared from Becker, Posselt, and Trénel, and in the following year papers from Behr on hysteria, and from Finkelstein and Scott on dementia. In 1894, Motet published a description of his own symptoms after being poisoned by fumes from burning charcoal, in which loss of memory and confusion of mind were prominent. In 1897, Jergolsky called attention to the frequency with which delirium of a transient character appears after gas-poisoning immediately the patient regains consciousness.

An important paper by Greidenberg appeared in 1900, entitled "Des Psychoses par Intoxication Oxy-carbonique," in which he fully described three cases of dementia which had come under his notice. One of the very few articles which have appeared dealing with disturbances of vision following CO poisoning was contributed by Purtscher in the same year. He pointed out that, in all cases where there are serious ocular disturbances, mental disorders, more or less severe, ranging from simple amnesia and confusion of mind to dementia, are met with.

In 1901, Truelle and Petit, in reporting an unusual form of loss of memory following CO poisoning, minutely examined the various forms of amnesia which follow intoxication by this gas. Later in the year followed the valuable contribution entitled "Des Troubles Intellectuels Consécutifs à l'Intoxication Oxy-carbonique," by Le Dosseur. In this work, Le Dosseur reported one of the most extraordinary cases of CO poisoning in the whole literature of the subject.

In 1902, two most important papers appeared by Hedren and von Sölder on the pathological changes in the brain and spinal cord; and Runeberg, about the same time, described an unusual case of mental disorder. In 1903, one of the most important articles on this subject, dealing in a very minute and thorough manner with the pathological changes in the brain, was published by Sibelius. In it and in the paper by Hedren, there are interesting plates showing the microscopical changes in the brain. The same year Croizet, writing on "Neuritis Oxy-carbonique," held, in many cases where paralysis, etc., followed CO poisoning, that the mental symptoms cannot be divorced from the neuritis, that they belong to what Korsakoff had previously called the polyneuritic psychosis, more commonly known in this country as Korsakoff's Syndrome.

Other important articles in which this subject is discussed have been published more recently by Panski, Engels, Revenstorf, Federschmidt, Lochte, Agostini, Stursberg, Knecht, Bloch, and Sanger Brown, the last-named contributing the only article we have chanced upon in American literature on this subject.

The only British authors, who have noted cases in which mental disorders were prominent, are the following, viz.:—Ross and Bury, who called attention to the most important and better-known cases recorded in the literature of the subject; Bury, who later reported a case of hemiplegia to the Commission on Compensation for Industrial Diseases; Scott, who published two interesting cases of dementia following poisoning by blast-furnace gas; and Sir Thos. Oliver, who described the mental disorders in two cases of poisoning by furnace-gas which had come under his own notice.

Having already described the peculiar mental conditions which may be found immediately after patients recover consciousness, it would probably be true to add that many minor cases occur in which the mental symptoms present are not sufficiently prominent to attract attention, more especially as these not infrequently pass off very quickly. In other cases, however, in which only minor manifestations of interference with mental processes are noted, these may become more and more exaggerated and last for a long time; may, indeed, even become permanent. We have described at some length, also, the remote mental effects which have followed poisoning by after-damp and the fumes from underground fires, all of which may likewise be found after poisoning by furnace, producer, and illuminant gases. From a variety of cases now to be described, the mental changes will be seen to show varied gradation, even to actual dementia.

The following is a case of our own observation which gives a good conception of the change in temperament sometimes seen after CO poisoning following "gassing" in collieries. This man was severely poisoned by furnace-gas, he was unconscious for nearly an hour, and the after-effects persisted for eight months. He had been a very healthy man, never before having suffered from illness. During the first two days after his accident, he complained of pain at the pit of the stomach, of severe vomiting, and also of great localised pain in the head, which felt, he said, "as if something were being driven into his head by slow degrees." He was off work for three or four weeks, when, although he had not quite recovered from these symptoms, he insisted on returning to work. For the first six months he continued to suffer from attacks of pain over the top of the head, making him feel, he said, "as if his head were going to open." He complained of indigestion and of loss of appetite, and his weight steadily decreased. Prior to his accident he had been good-

natured and easy-going, but for a few months after his accident, as his wife informed us, he was very irritable and suspicious, and lost his temper over trifles, which had previously been foreign to him. She also informed us that he had become very forgetful. He sometimes became quite apathetic, and instead of being continually active as he formerly was (being particularly energetic), he would sit still doing nothing and staring into vacancy for long periods. For the first six or eight months, although fairly regular at his work, he was continually ailing and complaining. However when he did begin to improve, his progress towards recovery was very rapid, his usual good spirits and alert mind soon returning. His memory, however, was the last mental defect to recover completely.

Although in many cases mental symptoms, such as loss of memory, may be present from the time the patient recovers consciousness, yet in others an interval of a few days may elapse before the mental disorder shows itself. But even in these cases, it will often be found that the patient on regaining consciousness shows cerebral symptoms, such as marked excitement and, it may be, delirium, or apathy and depression. This might be very transient, passing off in a few hours, and the patient may have had several days' absolute freedom from such symptoms, when suddenly he would exhibit pronounced mental disorder. That happened in a case, described in detail later, in which the patient, when he regained consciousness, was violently delirious. A few hours afterwards he had quite recovered and talked quite rationally, but on the third day his mind suddenly became a complete blank. This man had been exposed for a long time to the very high percentage of CO usually present in furnace-gas.

Von Sölder [1] reported one of the most instructive of these cases, of a man who was under observation in the hospital, and who was perfectly happy and healthy in the interval which elapsed between regaining consciousness and the development of severe mental derangement. In searching the literature on this point, we have met with a number of cases in which there was this interval of freedom from all symptoms; indeed, it seems to be almost characteristic of many of the cases which end fatally with symptoms of severe cerebral lesions. We have already called attention to the difficulty which this free interval creates in connection with cardiac and nervous conditions, such as neuritis, in cases which come up in Court for compensation.

The explanation of this cannot be looked for in the accumulation of the poison in the system. Sibelius[2] holds that the pathological changes

[1] Von Sölder. ; "Zur Pathogenese der Kohlenoxydlähmungen," *Jahrb. f. Psych. und Neurolog.*, 1902, Bd. XXII. p. 287.

[2] Sibelius : "Zur Kenntniss der Gehirnerkrankungen nach Kohlenoxydvergiftung," *Zeits. f. klin. Med.*, 1903, p. 3.

in the brain which he found furnish the solution. Owing to progressive changes in the vessels, the cerebral changes, which were at first slight, develop later into much more serious lesions with correspondingly graver mental symptoms. The changes in the vessels must, however, reach a certain stage before the destructive lesions set in; small hæmorrhages, thickening of the vessel wall, contraction of lumen, all leading to decreased supply of blood to the respective areas.

In many of the cases in which pronounced mental symptoms did develop, the poisoning had been severe, the coma profound, and in some cases violent convulsions had been present; but in a few other cases the poisoning had only been slight; indeed, in one or two cases on record the patient never lost consciousness. Relapses are common in these mental cases, for it is sometimes found that, while the patient is making rapid progress toward recovery, the symptoms return as markedly after he has experienced some unusual fatigue or mental emotion; for example, in Bourdon's case already reported (p. 261), in which for four or five days the patient lost his sight, speech, and hearing, he had a severe relapse following a fatiguing walk in the heat of the sun, three days after he had made very great improvement.

When men resume their work after they have been off for some weeks suffering from the effects of poisoning by CO, and during which certain minor mental symptoms have been present, the effect of mental and physical fatigue is often sufficient to bring about a relapse in their mental condition. In many of the cases of CO poisoning in which there have been marked mental disturbances, there is a remarkable tendency to remissions, the patient making steady and rapid progress to what might be thought an early and complete recovery, when suddenly there is a relapse. In a considerable number of such cases the second condition is found worse than the first. In some cases, although there are no decided mental symptoms, it is found that the patient is not so alert mentally as he had been, that sometimes under certain conditions he gets quite confused, that he is slower in thinking, and that his mental faculties have fallen to a slightly lower plane. In such cases it is found that the first mental condition to go is initiative, the patient losing confidence in himself and becoming much less self reliant. Indeed, in all cases in which mental symptoms are pronounced the higher cerebral functions are the first to deteriorate, and the first to be lost.

It has been found, further, that men who were formerly of a very stable and unemotional type of disposition become anxious, excitable, and nervous, and from time to time suffer from emotional attacks, from loss of control, and from fits of weeping and laughing. Sir Thos. Oliver in his *Diseases of Occupation* describes two cases, in one of which there was excitability and exaltation, and in the other, besides hysterical

excitability, a tendency to laugh immoderately at trifles and on the slightest provocation. In these cases the mental condition persisted for more than two years after the accident. Other patients who were formerly very active and intelligent became stolid, dull, and stupid. In others, again, there was profound depression and even melancholia. Greidenberg[1] holds that, in a large number of the cases, certain distinct phases in the mental illness can be detected. The patient at first becomes dull and forgetful, well-marked loss of memory then follows, and there may next appear incoherence and indifference grading to profound apathy, succeeded in some cases by great weakening of the mental faculties, in some instances even to the extent of dementia.

Attention may now be drawn in some detail to the most prominent mental conditions, such as delirium, loss of memory, and dementia.

Delirium, Disorders of Emotion, Acute Mania.

Attention has already been drawn to the fact that patients on recovering consciousness may be found to be in a very excitable condition, laughing immoderately, weeping, singing, shouting, or talking loudly in an incoherent manner, and behaving in many cases as if intoxicated. In a few cases the patient became maniacal. Paul Morceau, in his monograph "Des Troubles Intellectuels par le Gaz Oxyde de Carbone," called attention to the fact that in ancient history there exists a good example of certain intellectual disturbances so produced. Not far from the town of Delphi, on the slopes of Parnassus, was a grotto from which escaped an emanation which intoxicated the animals which grazed in the vicinity. There, was the oracle of Apollo, perhaps the most celebrated of all antiquity, more especially among those who owned allegiance to Greece. On a golden tripod, enveloped in this atmosphere emanating from the grotto, sat the Priestess of the God. Being very susceptible to its influence, probably from her nervous temperament, she soon became affected. Quickly she got into a most excited and frenzied condition, with face swollen and distorted, neck puffed out, and panting-breast, she offered a strange and awe-inspiring spectacle to the ignorant onlookers. Then rolling her head and neck about, waving her arms and shaking her whole body, she would begin to mutter and talk in a wild and incoherent manner. From her mouth would issue wild inarticulate cries and ravings, which the priests would collect with apparent care, the meaning of which they would translate according to their wishes. On analysing this poisonous atmosphere, it was found to contain a large percentage amount of carbon dioxide, as well as carbon monoxide and sulphurous acid gases.

Cases have already been recorded by Casper Liman and Jergolsky in

[1] Greidenberg: *Loc. cit.*, p. 63.

which, for a few hours after poisoning, the patient suffered from furious delirium and became almost maniacal. In some of the cases which have been reported, the patients were so violent that they required several men and the use of the straight-jacket to control them. This transitory mania, however, rapidly passes off, leaving in many instances no traces behind it. In two of the patients admitted to the Greenock Hospital after the Crarae disaster,[1] the transition from coma to consciousness was accompanied by delirium, which in one case was so violent as to necessitate the use of a straight-jacket.

The following, which was reported by Federschmidt,[2] is the most recently published case of transitory mania, and it has several points of interest. A merchant, 42 years of age, was poisoned by fumes from burning coal. His wife found him lying unconscious and breathing stertorously. The doctor had some difficulty in bringing him round. When the patient regained consciousness he suddenly became raving mad, sat up in bed, waved his arms in an erratic manner, and struck at everyone who came near him, crying, raving, and talking incoherently all the time. He tried to get out of bed, threatened his doctor whom he did not recognise, and later on fought, kicked, and struggled with him. His behaviour gave the doctor the impression of that of a man undergoing chloroform anæsthesia. In a few days the patient complained of vomiting and of numbness of the head, and for twenty-four hours after the accident he suffered from albuminuria. He made a perfect recovery. An important medico-legal point to be derived from this case is, that persons just recovering from CO poisoning may commit assaults and crimes for which they are not responsible. Seidel[3] says that persons who have survived CO poisoning, owing to their wild and suspicious behaviour, have been repeatedly charged with murder. In many of the cases, as in the above, this transitory mania passes off leaving no trace behind.

In the cases by Trènel, Bourdon, Kayser, Devergie, and others, where the mania appeared immediately after exposure to the gas or after the interval of a few days, or, as in Bourdon's case, where it almost disappeared for a day or two to reappear suddenly after a fatiguing walk in the sun—in all these cases it lasted longer. In one of Sir Thos. Oliver's cases, there was marked exaltation and excitability which persisted for two years. A victim, as in Devergie's case, may become quite sensible and be perfectly clear mentally, after he has wakened out of a deep sleep. In such cases there may be no after-consequences, except

[1] Fox: "A few notes on the symptoms and treatment of the victims of the Disaster at Crarae." *Lancet,* 1886, Vol. II., p. 725.

[2] Federschmidt: "Zwei Falle von Kohlendunstvergiftung in hygienischer. etc." *Muenchener Med. Wochensch.*, 20th July 1909, p. 1483.

[3] Seidel, in Maschka's Book on Forensic Medicine.

that the details of the accident and illness have almost entirely been erased from the patient's memory.

But in a number of cases the mental condition of the patient is found to be changed; from having been bright and cheery in manner he becomes strangely apathetic, stolid, perhaps depressed, and in some cases melancholic: nothing interests him, nothing rouses his attention. There is also loss of control over the emotions; the patient laughs or weeps on the slightest provocation. He becomes incapable of fixing his attention on anything for any length of time, then, later, unable to carry on the simplest routine work. Although in some cases the patient falls into a dull, somnolent condition, many others develop intense and most intractable sleeplessness. The following case shows the changes in the mental condition which come on after such attacks of delirium, etc. Leroy [1] quoted the case of a man, 28 years of age, who on recovering consciousness became wildly delirious, a condition which lasted for several hours, and was followed by a state of stupor and apathy which lasted several days.

It is rarely that the delirium is accompanied by hallucinations, acute poisoning by CO differing in this respect from chronic, but Ruata [2] called attention to the case of a man who had remained for a whole day exposed to CO and suffered much from vomiting and headache, who showed symptoms of acute delirium, accompanied by hallucinations, which lasted for five days. Bouchereau and Raffegeau reported a case where after CO poisoning a man became very agitated, restless, and excited, shouting and struggling so much that it required several people to hold him. In our case of complete loss of memory following exposure to blast-furnace gas, the patient immediately on recovering consciousness spoke in an incoherent manner and was violently delirious. A few hours later he began to talk quite sensibly, but three days after his accident his mind suddenly became a total blank. This patient, however, after a long time recovered.

It is not uncommon in mental diseases to find abnormal excitement among the first symptoms of mania passing into dementia; and so it is after CO poisoning; for although most of these cases make rapid recoveries, in others the patient passes into a state of dementia. Thomsen,[3] for example, reported a case where there was wild excitement, the patient struggling violently and shouting in an incoherent manner; this continued for two days, then disappeared to reappear five days afterwards, when the patient passed into a state of dementia. Scott [4] also

[1] Leroy, quoted by Le Dosseur. *Thèse de Paris*, 1901.

[2] Ruata: "Delirio allucinatorie acuto per ossido di carbonio," *Gaz. Med. di Torino*, 1892.

[3] Thomsen: *Berlin. klin. Woch.*, 1888, p. 675.

[4] Scott: Dementia resulting from Poisoning by CO, *The Lancet*. 25th Jan., 1896.

reported a case where a patient, after poisoning by furnace-gas, had convulsions for twelve days, and remained in an unconscious condition for fifteen days after the accident, when he suddenly became maniacal. This in course of time developed into dementia, and he died after eighteen months' retention in an asylum.

Loss of Memory (Amnesia).

Very careful consideration ought to be given to partial or complete loss of memory following exposure to CO, occurring as it does in a minor degree in many cases which recover from the acute poisoning. This mental disorder is much more common than some writers seem to indicate, and is a condition which until recently has been very little studied by writers on Forensic Medicine, some, indeed, contenting themselves with merely mentioning that it may occur, while others ignore it altogether.

In the great majority of the cases met with in literature, the loss of memory in CO poisoning has appeared suddenly, and has not been slow and progressive as in alcoholic amnesia, or as in traumatic amnesia following carriage and railway accidents, to which Brouardel[1] has compared it. Several times attention has been called to the fact that this loss of memory bears a close resemblance to that seen in persons who have been resuscitated after advanced asphyxiation by strangulation or hanging.

Marcel Briand[2] was probably the first to make a study of this mental condition. He quotes a number of cases to show that it is a common sequel of CO poisoning. In one of his cases, that of a young woman who attempted suicide by inhaling charcoal fumes, there was total loss of memory regarding the details of the accident and of a short period of time after it occurred. She was found unconscious, with a large burn on the left arm so severe as to expose the elbow joint. The neighbours found her lying against the charcoal stove. When roused, she was surprised to find so many people about her, but did not notice her terrible burn. She became much excited, first cried, then recited, and finally sang, gesticulating wildly all the time with her injured arm. She was removed to hospital, and on arrival there she explained that she had never got over the death of her grandmother which occurred six months before, that she had been inconsolable, and that she dreaded what the future had in store for her. She had no recollection of the journey from the house to the hospital, which took almost two hours. The attendant, who accompanied her, said that during the journey she refused to believe

[1] Brouardel; *Les Asphyxies par les Gaz.* Paris, 1896, p. 38.
[2] Marcel Briand: *Annales d'Hygiène Publique*, 1889, Vol. XXI., p. 356.

in her attempt at suicide. After three days she began, with the help of suggestion, to remember dimly that she had attempted to destroy herself by asphyxiation, but she had no recollection of what happened afterwards. Indeed, she held vehemently to her declaration that somebody must have thrown her on the fire, as what she intended to do was to suffocate not to disfigure herself. "If I had burned myself I would readily confess it, and I would remember it, as I recollect, now that you remind me of it, of having lit some coal." She was perfectly intelligent before the accident, and her memory was good. The latter was still good for events which happened previous to the accident, for she remembered perfectly a previous attempt at suicide by opening a vein which she made a fortnight before. On that occasion she was surprised by the neighbours. The loss of memory in this case, then, was incomplete for a few minutes previous to asphyxiation, for she remembered dimly her preparations for suicide; but there was total loss of memory of the details of the accident and of incidents for a short time after the completion of her act, as, for instance, during her journey to the hospital. Attention has already been drawn to similar cases of burns received in explosions in pits, where the victims had no recollection how they occurred.

In another case by Briand,[1] a woman, 62 years of age, formerly of a bright disposition, became melancholic, and fearing lest she should die in poverty, attempted to commit suicide by inhaling the fumes from burning charcoal, but was rescued. Some time afterwards she became very talkative, speaking incoherently. When questioned a few days later, it was found that her memory for events which had happened previous to her accident was perfect. From the hospital, where she remained four days, she was removed to the asylum, as she was still melancholic and had suicidal tendencies. When she arrived at the asylum, on being questioned regarding the motives which prompted her to take her life, she replied in a clear and precise manner, and gave a very exact account of the manner in which she had employed her time up to the moment she lit the stove. Briand lays great stress on this point, viz. :—that she had an idea that she lit the stove at the time of her suicidal attempt, but between the time of her accident and removal to the asylum, four days later, her mind was a complete blank. She could not say whether she had passed days, weeks, or months, from the time she was asphyxiated till she recovered from her illness, this space of time having been completely erased from her mind. Here, then, is a woman who previous to her accident had an excellent memory, and who was able to go through her household duties in an intelligent manner, under the influence of melancholia trying to asphyxiate herself; immediately afterwards her memory became defective, and after recovery it was found that a period

[1] Briand : *loc. cit.*, p. 348.

of time has been blotted from her mind. Certainly in this case, as shown by her attack of melancholia, there would seem to have been a predisposition to mental disorder.

This form of amnesia, where a period of time and the events immediately after the accident itself are absolutely forgotten, is very common, and many other similar cases could be cited. At the same time, there is a good deal of mental confusion which lasts only for a very short period, and which certainly does not help in any way the retention of memory, to the frequent appearance of which Artigalas and Magnan have drawn attention. Briand also points out an important medico-legal point in connection with this mental condition, viz.:—that this partial loss of memory in cases of attempted murder might prevent the intended victim giving evidence against the assailant, as he or she would not be able to furnish any particulars regarding the preparation for the crime. In other cases, again, a period prior to the accident may be erased from the patient's memory. In Sanger Brown's case, where a man of 34 was rendered unconscious for three days after illuminant-gas poisoning, the period of thirty-six to forty-eight hours previous to his accident had been erased from his mind. Although his memory for the early events of his life was good, it was only after much practice that he was able to remember his way about the house, and the names of his attendant and nurse. He was very emotional; he could read, but could not discuss current tropics. He died suddenly ten months after the intoxication.

Fallot[2] describes a case of retrograde amnesia in a woman of 63 years who attempted suicide by CO. Despite every effort, she could not recall to her mind the reasons which prompted her to attempt suicide or the details of that attempt. Likewise, she had forgotten what happened during the three preceding days, although she could recall having gone three days before her accident to the cemetery to pray at the grave of her husband, who had died a few months previously. She then returned home in a very sad condition, but had no intention of committing suicide. In other cases, however, the period of time lost is much greater. Take, for example, the case reported by Leudet[3] where, seven days after poisoning, the patient's intelligence had been incompletely restored. He could only recall his name and where he lived, but was neither able to give any explanation of the antecedents nor of the cause of his present condition. His mind remained in this condition for a considerable time.

Brouardel[4] saw a doctor who had been poisoned during the night by emanations from a stove in a room beneath his own. This man, after

[1] Sanger Brown: *J. American Med. Assoc.*, Chicago, 28th April, 1906.

[2] Fallot: *Annales d'Hygiène, etc.*, 1892, p. 244.

[3] Leudet: *Loc. cit.*, p. 523.

[4] Brouardel: *Empoisonnement par les Gaz.*, Paris, 1896.

being in a comatose condition for six or seven hours, was brought back to life. When he had sufficiently recovered to resume work, he found that he could not recall the names of his patients, where they lived, or the diseases from which they suffered. He had also forgotten the names and the doses of the drugs he was constantly in the habit of prescribing. Naturally his practice suffered and he lost patients every day, with the result that he decided to raise an action for damages against the proprietors of the house. Brouardel was called as an expert witness, and in this way became acquainted with the case. Brouardel, in remarking on the slow progress of the law in France, makes the remark that "our colleague had recovered his memory and his patients before the case came up, so he abandoned his suit."

In most cases memory comes back very slowly, but in the following case of Leroy[1] the return of the lost memory took place suddenly. A man, 28 years of age, after attempting suicide by charcoal fumes, was attacked with furious delirium and had to be removed to the asylum. Two days after the accident he was found in a condition of melancholia and stupor. The next day his wife came to see him, but he did not recognise her. On the fifth day he passed out of his stupor, but could neither give any account of why he was in the asylum, nor when he came there. He had no recollection of his attempt at suicide, or of his journey to the hospital. Next day, however, he became much better, asked where he was, and how he came to be there. He stammered considerably, but there was no aphasia. He now recognised his wife. He had no recollection of the events either following or immediately preceding his attempt at suicide, such, for example, as a letter he had written to his father. On the seventh day he suddenly regained part of his memory. Fifteen days later, he recollected his removal from the hospital to the asylum with two women and an attendant. But he had no recollection of the time which had elapsed from his loss of consciousness to the time of his departure from the hospital; that period had been completely blotted from his mind.

Following CO poisoning there is rarely loss of memory for events which occurred after the poisoning. In the discussion which followed the reading of Briand's paper, Beauvais[2] repeated the following case, which he had reported at a Congress at Turin. One day he received a visit from one of his confrères, who asked him to see his patients because he had been the victim of an accident. He sent Beauvais at the same time their names, their addresses, and his diagnosis of each case. Beauvais looked after the cases. After a certain interval of time his friend came back, asking Beauvais if it were true he had asked him to

[1] Leroy, quoted by Le Dosseur, *Thèse de Paris*, 1901.
[2] Beauvais: *Annales d'Hygiène Publique*, 1889, Vol. XXI.

relieve him, as he had no recollection of having done so. At the same discussion, Bouchereau[1] reported the case of a man and woman who were both poisoned by CO, the woman having had loss of memory for fifteen days; but the man, after an interval of a year, had not recovered his mental faculties, and remained insane.

One of the most constant features of this loss of memory is, that it is generally the most recently acquired knowledge which is the first to go, the memory of early events being left untouched. A patient, for example, will forget that he has just had a visit from his wife, or the time of day, although he has but a short time before been told, but may be able to give a long and correct account of what happened in his early days. Besides the total loss of memory for some definite period of time, it is not infrequently found that the general memory is rendered defective or impaired for a shorter or longer period. We have been struck by the fact that, on several occasions, the wife of a furnace-man or miner has volunteered the information that ever since her husband was gassed his memory has not been nearly so good. He cannot be trusted with the simplest messages, etc., owing to his being so forgetful. In some slight cases the patient forgets the names of his friends. In all cases, the names of unfamiliar objects are lost sooner than those of familiar things. The patient may have a perfect idea of what the name is, but he cannot find it, and the attempt to reach the phantom which is so tantalisingly near, makes him irritable. A good example of this is seen in Dosseur's case.

In some cases, as in that of Barthelemy and Magnan (p. 301), the patient suffered from paramnesia, recounting with detail things which had never happened, such as how he had been at one time walking about in Paris and at another time in his native town, which was purely imaginary. This mental phase is not infrequently met with in alcoholic neuritis.

The following is an interesting case we attended, in which the mental symptoms lasted for a long period, some, indeed, becoming permanent. A young man, aged 23, received a heavy dose of furnace-gas and was unconscious for some time, his fellow-workmen having considerable difficulty in bringing him round. On recovering consciousness he was in a curious condition, being excited, talkative, and raving in an incoherent manner quite unintelligible to his friends. His speech was changed in a remarkable way, being curiously blurred and of a gulping character. He was not a good speaker at the best, but for two or three months after being gassed one could hardly make out a word he said. Immediately after the accident and during the first day, he vomited a good deal, but this having passed away, he made no complaint of any

[1] Bouchereau: *Annales d'Hygiène Publique*, 1889, Vol. XXI.

kind. He was never at best very intelligent, but now he became absolutely stolid and stupid, and for the first few days could not even look after his natural wants. For three or four months his memory was almost a complete blank; and even after the lapse of a year, remained very deficient. The curious point about this case was, that after the first few days the patient's physical condition remained perfectly good, in fact, as he lived in lodgings and was only a labourer, he was persuaded to go back to work about eighteen days after he was gassed. We saw the man two years after this, when he appeared to be much brighter and more intelligent, but we were informed that he was much more stupid than he was before his accident, that his memory still remained very bad, and that his foreman had constantly to keep his eye on him while he was at work. To begin with, the man was badly educated, but he lost the little knowledge he had, forgetting even how to write his own name as he formerly could do. He was also unable to read, although prior to his accident he could do so with difficulty.

When dealing with the subject of susceptibility to CO poisoning, attention was called to the fact that certain people, owing in certain instances, probably, to heredity and nervous constitution, and in others to alcoholic habits, are initially predisposed to mental affections, hence exposure to the gas is apt to project into greater prominence the nervous sequelæ which follow the exposure. Rouillard[1] reports a case of a patient who was markedly predisposed to mental affections, both by her family history and by her own neurotic temperament. A woman, 47 years of age, was sent to an asylum owing to attacks of intellectual feebleness, marked diminution of memory, melancholic depression, incomplete knowledge of her acts, suicidal tendencies, etc. She was the daughter of a drunken father, and she had had sisters who showed marked hereditary stigmata, the first being a hydrocephalic polydactylous monster, and the second a girl who died of meningitis. The patient herself had had frequent attacks of hysteria about the time of puberty, in all of which attacks symptoms of a neurosis could be made out. She had had four full-time children and two miscarriages. Following a confinement when she was at the age of 46, she became melancholic and hypochondriacal, but her intelligence was unaffected and her memory good. She attempted suicide by inhaling fumes of charcoal. She had an attack of maniacal agitation which lasted for a very short time, but what struck her friends most was the fact that there was complete loss of memory. She recognised perfectly her husband and her two little daughters, but she was completely at a loss, when shown her youngest child of seven months. She denied very emphatically both her pregnancy and her confinement; she had forgotten all about a trip she made with her

[1] Rouillard: *Essai sur les Amnésies*, Paris, 1887, p. 169.

husband a few days before her attempt at suicide; she could not tell her age; she did not know the year of her birth; she had completely forgotten the ages of her two daughters; she could not say what day it was; she did not know when or how she came to the asylum. During the next three months the patient became less melancholic, her appearance was better, she was not nearly so dull, and it could easily be proved that the stupor and mental disorder found from the beginning were due to the blunting and confusion of the mental faculties and not to dementia. Her memory was still very poor; she did not know her own bed; and some hours after a visit from her husband she had no recollection of it. Later, her mental condition became very much improved, her memory especially making marked progress. At the time of writing these facts, although the patient was more or less forgetful, she was able to recall all the events which had taken place during the few months following her attempt at suicide.

In the following case, which is minutely and carefully described by Trénel,[1] the patient was also markedly predisposed to mental disturbances, A dressmaker, 21 years of age, with a bad family history, had signs of mental degeneration, suffered from melancholic depression, and was subject to fits of hysteria. She attempted suicide by inhaling charcoal fumes. There was complete loss of memory for everything at the time of her attempt. When her mother told her about it, she had no recollection of the fact; indeed, she doubted the truth of it, saying: "I have been told about it, but perhaps it is not true." A period prior to her attempt at suicide had also been erased from her mind. She did not, for example, remember when she had ceased working owing to an illness, nor had she any recollection of that illness. *A period following her attempt at suicide was also blotted from her memory*; for example, a long walk she had taken with her father, the day after she was taken to the asylum, was completely forgotten. Although she understood perfectly that she was in the asylum, she lost her way in the wards, did not know her own bed, and did not recognise the inmates of her ward. When outside the asylum, she could not find the entrance through which she had just come. She had only a vague idea of time, and could not say whether it was July or August. As in nearly all these cases, her memory for old events remained perfect. For a long time her mind was confused owing to the defect of memory, for she could never remember for any length of time what she had been told. It was difficult to fix her attention, and the simplest information imparted to her was forgotten almost at once. When she was dismissed from the asylum she was still very forgetful.

The following, a case of continuous amnesia following CO poisoning

[1] Trénel: "De quelques Symptomes consécutifs à l'intoxication aiguë par l'oxyde de carbone." *Gaz. Hebd. de Méd. et Chir.*, 1895, p. 351.

was reported at considerable length by Truelle and Petit.[1] It contains many interesting and instructive features. A man, 55 years of age, of a bright and cheery disposition, was so much affected by failure to get work that he became depressed almost to melancholia. Before attempting suicide, he wrote from Paris to his children in Brussels telling them of his resolution. He was found unconscious in bed, and was taken to St Louis Hospital on the 9th March, 1900. He regained consciousness on the 10th. He was dull, confused, and apathetic, and had no recollection of his attempt at suicide. The knee-jerks were notably diminished, but there was no motor or sensory disturbances. Indeed, the authors lay particular stress on the point that the amnesia was uncomplicated by any other mental phenomenon. His speech was monotonous, deep in tone, and a little loud. Cerebration was slowed, the patient only replying to a question after thinking for a little time. His emotions were blunted, he was easily led, and was usually quite quiet and passive. Apart from this slight intellectual depression, there was nothing unusual about the mental condition except the loss of memory. He had no recollection of his attempt at suicide, or of the events which preceded it for a certain time, the boundary of which was ill-defined. He remembered stopping work on the 6th of March, recalled after a little hesitation the street in which he lived, but could not tell the number. The visual memory of his dwelling was quite good, as he knew the number of the rooms, and could describe the furniture and the colour of the wall-paper. Speaking generally, he most frequently forgot names and dates. This retrograde amnesia, with irregular intensity, had a bearing, then, in a confused way and for an ill-defined period, on the events prior to his attempt at suicide, but certainly did not stretch over a long period. Memory for the past was perfect. He could give a detailed account of the whole of his early life. The patient's memory was also in a curious state of fluctuation; for example, he would one day give the correct number of his street, then the next day he would forget it; he could not recall during the day the name of a companion which he had remembered in the morning; and so on. He had absolutely forgotten the events which preceded his attempt at suicide, and the letter he wrote to his children. "No," he said "I have never attempted to kill myself. I know only this that I had black thoughts at not being able to find regular work, and being in straits." He had no recollection of his stay in St Louis, of his departure from there, nor of his passage from there to the Infirmary.

All the time the case was followed by the authors till 18th January 1901, when the patient was transferred to the Asylum at Tournai in Belgium, this loss of memory persisted. As a curious fluctuation was

[1] Truelle et Petit: "Un cas d'amnésie continue, consécutif à une tentative de suicide par l'oxyde de carbone," *Archives de Neurologie*, Paris, 1901, p. 86.

found in the extent of his loss of recollection of events, etc., prior to his suicidal exposure to the charcoal fumes, so there was during these nine months which followed, for there were great differences in his ability to remember past events and to acquire fresh knowledge. Even the most rudimentary facts had to be repeated many times before they were fixed in his mind. His idea of time, also, was very deficient : thus he said that he did not remember having had a conversation with the doctor the day before ; and two days after he was brought into the hospital he said he had been in eight days. A week after his wife had been to see him, he thought he had seen her that morning. He could not recall to-day what he had eaten yesterday, and when he was taken to his dormitory he could not find his bed. He was made to write his name and age, but five minutes afterwards he had no recollection of having done so, in fact did not believe he had written what was shown to him. He read an article in the paper about the Transvaal War, and when he had finished it, he knew that it was about the Boers, and that was all. He lit a cigarette many times a day in the hall, notwithstanding that each time he was told that smoking was not allowed there : and such things as these went on daily. Memory for automatic actions such as walking, manual exercise, professional work was good. He could write and speak quite well. After ten months there was slight weakening of the mental faculties, bearing on the loss of memory. But the unusual feature of this case was the continuation of the loss of memory without any other mental disorder; for it is generally found that where this has persisted for any length of time, mental symptoms such as gradual weakening of the mental faculties, even progressive dementia, are usually present. In this case, immediately after consciousness was regained, there was a certain amount of mental confusion, specially characterised by apathy and by slowness in cerebration. The authors thought that the loss of memory which preceded the attempt at suicide (such as the name of his late master, number of street, etc.), should be put down to the mental confusion which is so frequent in such cases as to be almost characteristic of CO poisoning, seeing that memory could at times be awakened, and re-vivified.

Artigalas[1] reported another interesting case where it was found that, although memory was very defective, the patient still retained her sound judgment. A woman, aged 55, who had previously attempted suicide at 14 years of age and twice thereafter, resolved with her husband to commit suicide, as they were in misery. The man was found dead, and the woman, after great difficulty, was resuscitated. There was no aphasia. The left pupil was found markedly contracted, the right normal. She was sent to the asylum, where Magnan was surprised to find that although there was considerable weakness of the memory, her reasoning

[1] Artigalas : "Des Asphyxies Toxiques," *Thèse de Paris*, 1883, p. 92.

powers were quite unimpaired. She appreciated things at their true value, but forgot all about being visited by a friend the day before. She could not find her way about the house, did not remember the date of her entrance into the asylum, and thought it was 1870 or '71 when it was 1881. Agostini[1] has also reported two cases where there was marked retrograde amnesia. The following case by Bouchet[2] is reported by Cacarrié, where a woman, who was an epileptic with a tendency to melancholia, attempted suicide. She was found unconscious, with her child dead beside her. Late next day she was removed to the asylum. The only word they could extract from her when repeatedly questioned was "partout." She had forgotten all about her attempt at suicide. She remained in a dull, dazed condition for a few days.

A case, where the loss of memory was associated with melancholia but without any other mental disorder, was reported by Greidenberg[3] in his interesting article on CO psychoses. A woman and her son, a strong young man, were poisoned by the fumes from a stove. The son, who was in a state of alcoholic intoxication at the time, and therefore most susceptible to CO poisoning, died. The mother was found in an unconscious condition, and remained comatose for a week. On regaining consciousness, she complained of great headache and pains in the chest, and became melancholic and lachrymose. Later, her speech became disordered. She remained in this condition for two months. She was quite clear mentally, and could give a detailed history of her past life up to the time when she went to bed on the night of the accident. After that, well-marked blanks were found in her memory. For example, she thought she had only been ill for five days, whereas her illness had lasted more than two months. She understood she was in hospital for treatment of incontinence of urine. During her stay in hospital she was once attacked by delusions, but these disappeared the following day. Although her memory for recent events still remained poor, her mental state otherwise had so much improved that she left hospital. Commonly speaking, where melancholia is found, it is usually associated with other mental disorders, such as confusion. As Dosseur has pointed out, in the large majority of cases it is the result of the mental state prior to the poisoning by CO; indeed, it is generally this in nearly all these cases which leads to attempts at suicide.

As all knowledge is based on memory, when memory is completely lost, all that the patient has learned—every thought, word, and action—must go, and he has to learn everything over again, even how to speak, how to read, etc. The aphasia, then, in such cases is the result of the

[1] Agostini, quoted by Sanger Brown: *J. American Med. Assoc.*, Chicago, 1906.
[2] Bouchet, quoted by Cacarrié: *Essai sur les Amnésies Toxiques*, Paris, 1887.
[3] Greidenberg: *Annales Médico-Psychologiques*, 1900, p. 66.

loss of memory, and is well described as amnesic aphasia. The most exaggerated form of this existed in a case which came under our observation. When this loss of memory has lasted for some time, it will lead to deterioration of the other faculties, the operations of which, of course, are impossible without the help of memory. Certain of these cases end in dementia, but in the following case it was surprising how little damage was done to the mental faculties, notwithstanding the almost complete loss of memory.

The most remarkable case of CO poisoning which has come under our own observation—one of the most remarkable, indeed, in the whole literature of CO poisoning—occurred about fifteen years ago in Coltness Ironworks. It has been alluded to by Dr Scott when reporting a case of dementia, which we shall describe later. An engineer, 20 years of age, who is described as being a very clever and most intelligent workman, was sent into an exhauster to do some repairs about the fans. It must be explained that in these exhausters there is just room enough for one man. The air inside was certainly tainted with "clear" gas. After finishing what he had to do, the young man came out, and although he complained of feeling a little giddy, there did not seem much wrong with him. He had only been outside a few minutes when he remembered he had left his chisel inside, and as this would have damaged the blades of the fans he went in for it. He had no sooner entered than he collapsed, and sank unconscious to the bottom of the exhauster. There was no way of getting him out except by taking the exhauster to pieces, unscrewing bolts, etc., and by the time this was done, the unfortunate lad had been about an hour in this confined place, breathing an atmosphere with a certain percentage of CO. No one apparently thought of getting oxygen and thus purifying the air of the exhauster. Nobody expected to get him out alive, and it was only after a long struggle and by hard work that Dr Millar, who had now arrived on the scene, was able to bring him round.

As to the further progress of the case, most of the following particulars were obtained from an intelligent engineer who sat up with the patient for the first four weeks, and who, living as he did next door, had abundant opportunity of seeing his progress from day to day. The accident happened between eleven and twelve o'clock in the forenoon. The patient, after resuscitation, was dazed and stupid, talked in an incoherent manner, and was very "heady" till five or six in the evening, when he began to talk quite rationally, and was able to write his name on paper when asked to do so. These points are important in view of what happened afterwards. One may say, then, except for the first few hours after being gassed, that he continued for nearly three days with very little apparently wrong with him either mentally or physically. Suddenly,

however, on the third day his mind became a complete blank. He knew nobody, not even his own mother, and he could do nothing on his own initiative. Although he was never at any time outrageous or difficult to control, he required to be very carefully watched, as one never knew what he would do next: but he was easily guided. He knew sufficient to rise to urinate, but once on the floor he had forgotten where he was, and passed his urine against the wall or bed. His powers of speech, sight, and hearing were good. If asked to look through a window at a horse and asked if he knew that it was a horse, he would repeat the word horse, but if questioned further, his answers would be silly and irrelevant. During the first few days of this mental condition, the only thing he would say was "Hoh! Mother," and this he would shout at the pitch of his voice every little while and for no evident purpose; and although his mother was constantly with him in the room, standing in front of him while he shouted, he did not recognise her or appear to understand why he shouted for her. In short, his mind was a complete blank for days.

This went on for a month or two, when there was a slight improvement, but even at this time, if shown his trousers and asked what they were, he would take them up and look at them in a dull fashion, and if asked to put them on, he would not know how to do so. If shown a bunch of keys, although he had a glimmering of an idea that they were for opening something, instead of going to the chest of drawers which stood beside him, he went to the wall and fumbled about with them. He was described as being at this stage like a big simple child who was very easily managed. For months he was led about, and his mind had to be opened up like that of a child. After a time he became a great deal better, and one of the teachers in the village school gave him lessons. His whole training at school, and at the night school during his period of apprenticeship, had been completely erased from his mind. He had forgotten everything, even the most elementary facts, and how to read and write; indeed he had to begin like a child at his alphabet, and his progress was very slow owing to his memory being so defective. After he had gone a certain length in his studies, he went again to the night school. Soon after this he made a start at his work, but here also he had to begin his apprenticeship over again, as he had forgotten everything about his work, even how to handle his tools. There was, therefore, in this case loss of memory for automatic actions, differing from the case of Truelle and Petit where this form of memory remained. Gradually however, he improved, and about eight years ago he married and has now two children.

His condition in 1910 was thus described:—He is a tall, powerfully-built man, in the best of health. He talks rationally and intelligently, and it is only those who knew him before his accident, or pay very

close attention to him, who can say that there is anything out of the ordinary about him, so sensibly does he behave. His foreman and those working with him who knew him before his illness, declare that his mental capacity is only a shadow of what it was. If properly directed he is a good workman with his tools, but when given work which requires initiative he is of little use. He requires to have his work carefully pointed out to him and explained, and he will then work well. Whenever he has any responsible job to do, he is sure to get excited. His memory is still very bad. He is restless, what the men call "fidgetty," and very easily excited and put about. Although very good-tempered and easy-going as a rule, he seems to lose command of himself, becoming angry and excited. In fact his fellow-workmen for a time were very chary about annoying him, as they realised that when roused he was not responsible for what he did. After attacks of anger or excitement his memory becomes much worse. He is often preoccupied; stands quite still staring into space for a considerable time; after which he will quietly resume his work. He frequently misunderstands things, and it takes time to enable him to arrive at a proper understanding of what is meant. When he started work again he was childish and sometimes smiled in a vacant way, but this has quite disappeared; indeed, one might speak to him for a considerable time without noticing anything peculiar about him. He is still improving. This case illustrates, therefore, how a patient's memory may remain defective for months or years, and yet how by education the mind may be developed afresh. In other cases, as in Trénel's, it was only by mnemonic exercises that memory was improved. With regard to the awakening of memory, Briand wrote—"The patient learns to remember in the same fashion as certain aphasics learn to speak. But the awakening of the memories is incomplete, and certain parts of the scene remained enveloped in a mist."

Dementia.

A considerable number of cases of dementia following CO poisoning have now been reported, and attention has already been drawn to cases where this, associated with other mental symptoms, was present.

When we remember certain cases of hemiplegia, etc., in which there were gross lesions in the brain, and which have been already described in the chapter on paralysis, we need not be surprised to find this condition accompanied in some cases by mental disorders.

Bourdon [1] was one of the first to call attention to dementia following CO poisoning. He quoted a case by Ferrus, [2] where in a young man

[1] Bourdon: *Thèse de Paris*, 1843.
[2] Ferrus: *Gaz. Méd.*, 1836, p. 715, quoted by Bourdon.

general paralysis with dementia followed asphyxiation by charcoal fumes. It will also be found by a study of gas-poisoning that many of the cases of dementia are of the general paralytic type, and that a considerable number of them are found, as we should expect, in persons over fifty years of age, because serious cerebral lesions are more probable at that age, owing to the condition of the blood-vessels. For example, Laborde[1] reported the case of a man of sixty-seven who, after CO poisoning, was suddenly attacked by dementia. Bouchereau and Raffegeau[2] also reported a case where it set in suddenly in an old subject. An old couple attempted suicide by asphyxiation (p. 268). The man, 67 years of age, was most seriously poisoned, being unconscious for several days. His whole mind was dulled and confused, and his temperament completely changed. He was formerly a most intelligent man, but he now became incapable of the least mental effort, his only desire seeming to be to satisfy his physical needs. From the very commencement in this case there was dementia. Bizarre, delirious ideas were also present. He took his wife for his sister, and spoke of his recent marriage with a cousin. After his accident, from being a thin and puny man he began to put on flesh. There was also loss of sight in this case, a careful examination revealing a considerable degree of interstitial neuritis of both optic nerves.

The following case of Sibelius[3] presents similar features. On 30th May, 1901, a workman, 20 years of age, who had always been healthy, tried to dry a cistern near a coke fire. After only five minutes' exposure to the fumes he was found unconscious. The following day he regained consciousness, when it was found that he was completely blind in both eyes. On the sixth day of his illness he had convulsions, and again became unconscious; he remained in this condition for four days. The pupils were dilated and did not react to light, and there was a passing paresis of the left facial nerve. At frequent intervals he had convulsions, especially in the right side, and continually in the right arm and hand. The neck was slightly stiff, and there was paresis of the left side of the body. There was fever which persisted for four days, when the temperature returned to normal and he regained consciousness. It was with great difficulty that he could answer questions, and he spoke in a disconnected fashion. Occasionally he had fits of anger and quarrelled with the other patients; at other times he would weep and become melancholic Sometimes he had attacks of acute delirium with hallucinations of sight Both legs became paretic. He could not stand, and even when lying in

[1] Laborde, Les Poêles Mobiles, *Bull. de l'Acad. de Méd.*, 1889, p. 531.

[2] Bouchereau and Raffegeau: *Loc. cit.*

[3] Sibelius, "Zur Kenntniss der Gehirnerkrankungen nach Kohlenoxydvergiftung," *Zeits. f. klin. Med.*, 1903, p. 3.

bed he could not move his legs with any freedom. From time to time his mind would become a little clearer, but he gradually drifted into a condition of dementia. After the second week of his illness, a large bedsore developed over the sacrum. Two months after the accident he was seized with convulsions, after which his condition gradually became worse. His temperature rose to 39°C. a few days before his death, which occurred on 1st September. The ocular symptoms persisted to the end. The blindness and mental disturbances in this case were explained by lateral areas of softening in the occipital lobes and in other parts of the brain.

We have already called attention to the fact that where certain mental defects, as loss of memory, last any length of time, grave deterioration of function, even to the extent of dementia, are almost certain to be encountered. In many of the cases there is a preliminary stage of excitement, followed by a quiet period and mental deterioration. In Thomsen's case, a man of sixty-four, after CO poisoning, was for twenty-four hours unconscious. He rallied, but complained of being quite worn out and of persistent headache. From this he recovered for a short time, but a fortnight later the acute symptoms returned, he became delirious, and suffered from mental confusion and hallucinations. Later, there was rapid enfeeblement of the mental faculties, and he fell into a state of dementia. In this case, as in many others, there was great enfeeblement of the muscular force, with staggering gait and exaggerated reflexes. The speech was slow and monotonous.

Cramer[1] reported the case of a woman of 71, which is interesting because of the lesions found at the autopsy. These we shall have occasion to discuss further. This patient was a healthy woman, and her family history was good. She was found unconscious in the morning poisoned by CO, and remained in this condition for three and a half days. Then consciousness returned little by little, and she complained of great weakness. Eight days afterwards she became delirious, spoke much nonsense, raved, and scolded her friends round about her, and at the same time was very confused mentally. She suffered much from sleeplessness. By and by she became completely apathetic. The pupils were widely dilated and reacted sluggishly to light; the eyes were half-closed. The whole body was found to be slightly hyperæsthetic. When loudly spoken to, she seemed to understand a little of what was said, and replied by movements of the head. The face was expressionless, but there was no paralysis of any part. Three months later she had exacerbations of fever, the temperature, however, never being more than 39°C. She became very restless, her limbs were continually moving, the

[1] Cramer: Anatomischer Befund im Gehirn bei einer Kohlenoxydgasvergiftung. *Centralbl. für Allgemeine Path.* July, 1891, p. 545.

movements of her arms simulating the act of embracing. The cardiac weakness which was present in a marked degree from the beginning, gradually became more pronounced, and she died.

While in a considerable number of cases of dementia there is a preliminary stage of delirium, excessive excitement, restlessness, and sleeplessness, in others the opposite conditions, depression, apathy, and somnolence may be experienced. Gnauck[1] has reported a case where this preliminary mental phase was found before actual dementia set in. A girl of 23 years was so seriously poisoned by CO that she remained unconscious for several days, and in a very somnolent state till the eighth day, when there followed a stage of deep apathy, the patient being dull and dreamy and difficult to rouse. A month after this she was seized with severe headache and backache, and again lapsed into a somnolent state, passing urine and fæces involuntarily. This gradually merged into a stuporose, dreamy state, with marked loss of memory and dementia. At the end of five months, however, she began to improve, her mind gradually opening up.

The following interesting case of dementia following CO poisoning, one of the very few recorded in English literature, has been reported by Dr Scott,[2] who has had considerable experience of gas-poisoning cases. A workman at the Clyde Ironworks was found in an unconscious condition poisoned by "clear" gas, which contained 25 per cent. CO, from one of the flues in connection with the ammonia work, where he had been working. After great trouble, medical help had just managed to bring him round when he developed violent convulsions. These continued at intervals for almost twelve days. He remained in a semicomatose condition for fifteen days after the accident, when suddenly he became maniacal. He was sent to the asylum, where his condition, which had now developed into confirmed dementia, got worse, and he died after being eighteen months in the institution.

In some cases, notwithstanding the apparently serious nature of the mental disorders, the patient may make a rapid recovery. Finkelstein[3] reported the cases of two workmen who were poisoned by gas while repairing a gas-holder. One died. The other was brought out in an unconscious condition which lasted three days. He then became so well that he was sent home, but in a very short time he showed signs of psychic disturbances, which became more and more severe till he had to be sent back to hospital. Usually these mental disorders, exemplified by

[1] Gnauck: *Charité. Ann.* 1883, p. 409, quoted by Koch, loc. cit.

[2] Scott: "Dementia resulting from Poisoning by CO." *The Lancet*, 25th Jan., 1896.

[3] Finkelstein: "Dementia Acuta in Folge von gaz-pauvre-vergiftung," *Jahrbücher f. Psych. u. Neurologie*, 1896, Bd. XV. p. 116.

loss of memory and confusion, gradually become worse, till acute dementia with stupor supervenes. In the course of this psychosis there were signs of paresis of the left facial nerve, which disappeared as he got better. The patient made a rapid recovery, but it was found that he had no recollection of the events which had happened for a few hours previous to his accident, or of the illness which followed.

In some cases, as we have already pointed out, there may be a quiescent period after the acute symptoms have passed off, the patient being apparently comparatively well. Hedren [1] reported such a case in which the patient felt well for a short time after recovery from the acute symptoms. Then followed headache, shooting pains in the limbs with gradual paresis, staggering gait, incontinence of urine, exaggerated knee-jerks, muscular rigidity, fibrillary tremor and spasms of muscles, gradual loss of memory, and a peevish, childish condition which passed on to complete apathy and dementia. This was terminated by an acute attack of broncho-pneumonia. Huchzenmeyer [2] reported a case in which, ten days after CO poisoning, intense neuralgia developed in the upper part of the thigh, and continued fourteen days. The patient had greatly improved at the end of three weeks, when suddenly mental disorders developed, exhibited by restlessness, excitement, hallucinations, and mental confusion.

We have on several occasions referred to a case by Barthélemy and Magnan,[3] which presents so many interesting features that we think it necessary to record it at some length. The patient was brought into hospital on the 17th April in a perfectly unconscious condition, with generalised convulsions. When he was brought round, it was found that he could understand nothing, that he moved very little, and that there was very little sensation. The eyeballs were agitated with convulsive movements, either vertical or rotatory, these movements being so pronounced as to be perceptible under the closed lids. When the eyes were examined, they were seen to move from within outwards and from without inwards with rhythmic movements. There was no nystagmus. There was retention of urine which lasted for five days. No sugar or albumen was present. For two days the muscular contractions continued, at one time feeble, at other times so powerful that two attendants were required to hold him down. On the morning of the third day he groaned from time to time, and appeared to have severe pain in the chest. There was notable *hyperæsthesia* of the skin, the slightest pinch causing

[1] Hedren; "Zur Kenntniss der nervösen Nachkrankheiten bei akuter Kohlenoxyd-vergiftung, nebst einigen Bemerkungen über ihre forensiche Bedeutung," *Nordiskt. Med. Arku.*, 1902, No. 20, p. 1.

[2] Huchzenmeyer, "Ueber Kohlendunstvergiftung," *Inaug, Dissert,*, Berlin, 1866.

[3] Barthélemy et Magnan: *Annal. d'Hygiène Publique*, 1881, Vol. VI., p. 407.

him to groan and tremble. He was quite blind. The same night he spoke a few words and, although during the night he was very restless, the convulsions were less frequent. Next day he spoke much better, but he had no recollection of what had passed, and in estimating the length of his illness, perhaps from his feebleness and from a feeling of great fatigue and faintness, *he thought he had been ill for seven or eight months.* The convulsions became less frequent, but a certain amount of muscular rigidity remained, so that it was impossible to bend his arms or legs. He now understood all that was said to him. He was not able to raise himself in bed; and if he was lifted slightly, his head would fall on his shoulder. The spasms and fibrillary contractions of muscles now disappeared, but there was still a general trembling of the limbs, which were very weak. On the fourth day there was a slight attack of bronchitis.

By 22nd April, intelligence was still dull and his thoughts still wandered. He had no idea where he was, could not recall what he had done, and thought he had been in hospital only since the morning. He thought that the year was 1875 and that he was in his native town, although in reality he was in Paris. He passed urine and fæces involuntarily. The next day a very profuse papular, acneform eruption, outlining the position of the lungs, appeared on the chest, also round the nostrils and on the nose. He could not pass urine. On the 25th April, eight days after the onset of illness, he was still in a state of complete dementia, and his ideas and thoughts still wandered. Although in bed, he assured the doctor that he had just arrived from Paris, and he gave a description of what he had been doing there. The next day he believed he was in Paris and not in the hospital. He was quite unable to say where he was when he arrived and what brought him there. He had still the same intellectual impotence and incoherence in speech. He spoke freely and replied quickly, his articulation of words was perfect, but the answer would be quite wrong and quite devoid of sense. He was quite lost as to time, did not know the year, and had no recollection of his accident. His sight was very limited. He did not suffer any pain, but was feeble, clumsy, vacillating, hesitating, without energy and without confidence. He sat up with difficulty in bed, but could walk if he were supported.

A month after the accident, he had regained to a considerable extent his mental and physical faculties. There was no delirium, and he now knew where he was and why he had come there. He was sometimes capable of reading, but he forgot a few minutes afterwards what he had read. Two months after his accident he attempted to resume work, but had to give it up owing to mental weakness. Seven months later his memory was still very feeble. He could not tell what day it was, nor had he any idea how long he had been in the hospital. He was emotional. It was

with the greatest difficulty he was able to move about the streets; as he was so easily bewildered and confused, for, as he said, "at certain moments I remember nothing." There was slight paresis of the left side of his face.

The progressively continuous changes in the mental condition are well shown in the following case recorded by Gnauck.[1] The patient, aged 56, had always been healthy and most intelligent; the family history being good. When poisoned by illuminant-gas, he was unconscious for twelve hours. On recovering, a distinct change was noted in his mind. He became quieter and apathetic, spoke little, kept to himself, and began to read his Bible constantly. His memory became weaker. Later he became more peculiar, more depressed, ate little, and was very sleepless, sometimes refusing to go to bed. He did not go to work or, when he did, he was constantly doing stupid things, as, for example, watering the flower-beds during the rain. He became restless and suspicious, putting all his money in his pockets, and when asked for some, said he had none. As his excitement increased, he was taken to the asylum. When he came there he was very quiet, appeared frightened, and gave the impression of intentionally concealing his thoughts. His memory became worse, although it had been very good up to the time of his accident. He now complained of pressure in the head, but there was no motor or sensory disturbances. He became more approachable, and during his last year occupied himself much with religious matters. He thought angels bent over him at night, but he would not say whether or not he heard real voices. He became very restless and afraid, and consequently was not able to sleep. Shortly before he died his mental condition changed very much. Instead of lying in bed, he kept moving about. He looked excited, but slept well, ate heartily, and declared that he was in a new world. He remained like this for a few days. His walk latterly had changed very much; when he tried to walk, he would fall forwards. Latterly he could not stand, and fell back towards the wall. He died suddenly three months after being taken to the asylum.

Greidenberg,[2] in 1900, contributed a valuable article on the psychoses following CO poisoning, in which he reported the following cases:—

Acute-Dementia with trophic disturbances. Woman of 58 years was poisoned during the night by the emanations from a stove in her bedroom. The next day she woke up with intense headache and nausea but soon recovered, although for the next few days she complained of headache and weakness of the legs. Then little by little a state of marked depression developed. She became sad and taciturn. She was a strong, healthy-looking woman, but her expression was depressed and

[1] Gnauck: *Charité. Ann.* 1883, p. 409.
[2] Greidenberg: *Annales Médico-Psychologiques*, 1900, p. 58.

apathetic, and she kept an obstinate silence when questioned. She refused nourishment, and even offered resistance when she was forcibly fed. Five days after her accident she was seized with severe diarrhœa, with a considerable amount of blood in the motions; in the course of the day a generalised pemphigus appeared all over the body, the temperature rose to 39°C, and the pulse became very weak. Large dark violet spots were found dotted over the skin in different parts of the limbs and in the lumbar region. The diarrhœa became worse and dysenteric in character, and she died six days after her accident. Greidenberg regarded the case as one of acute dementia following CO poisoning. The second case we have already described under loss of memory (p. 294).

The third case he regarded as one of paralytic dementia, a number of cases of which have been described in literature as following CO poisoning, asphyxiation by hanging, and poisoning by various toxic agents. A man of 45 years of age tried to asphyxiate himself. He was found unconscious, and remained in that condition for twenty-four hours. When he came to consciousness he complained of extreme and generalised muscular weakness, but during the next ten days he made such improvement that he resumed his work. His friends, however, commenced to find him strange in conduct, he was absent-minded and forgetful. For example, when he wished to make a cigarette, he would roll the paper but forget to put in the tobacco. He also got quite muddled and confused in his work. He had frequent attacks of great restlessness and distress without any apparent cause; sometimes he got very much excited and suffered much from sleeplessness. A doctor was called in, who found that the patient's answers to his questions were quite incoherent. When asked his age he replied 33, and when asked how long he had been married, he also replied thirty-three years. A month after his accident his condition became worse; the weakness of memory more exaggerated; his attacks of excitement more frequent; and he was sent to the asylum. When he stood up, he trembled so much that it was with the greatest difficulty he could keep himself erect. There was paresis of the right side, and the right eye was more open than the left. The tongue was drawn to the right side, and was markedly tremulous. The pupils were unequal. His gait was uncertain like that of a paralytic. There was an impedient in his speech, certain sounds were pronounced in a very indistinct manner, and he often omitted the last syllable of words. His replies to questions were often quite incoherent and wrong. He had no idea why he was there, or how or when he had come. He was now quite indifferent to everything. Six weeks after his accident he became delirious, suffered much from loss of sleep and was very restless, speaking and laughing all night. At this time he could give a very good account of his past life, but he could not recall recent events or why he was in the asylum. The day after, when

asked his age, he said 12; and he did not know the date. During the next fortnight his condition improved very much. Then he had another attack of mental confusion with incoherence of thought and speech, great weakening of memory, and restlessness. He declared that his brother had been put in the same room as himself, and when asked to point him out, he stepped to a bed indicating a patient to whom he had never spoken. Two or three days after this his mental and physical condition improved very much, he put on flesh, but there was still mental confusion and weakness of memory. Nine weeks after his accident his paralysis had entirely disappeared, but profound apathy and indifference to what was going on around him still remained. A fortnight later his memory began to improve, and he gradually recalled events which he had previously forgotten. He remembered the date of his entrance into the asylum and the length of time he had been there, but he had still the delusion about his brother being in the same ward. Three months after his accident his mental condition had greatly improved, he began to interest himself in his surroundings, asked many questions, and now began to speak of leaving the asylum, which he did in a few days.

Musso [1] was one of the first to describe cases which were very similar to that of Greidenberg. He described five cases of what he called pseudo-paralysis following chronic CO poisoning. Under this name, pseudo-general-paralysis, a large number of cases have been described, following poisoning by syphilis and alcohol, lead, morphia, bisulphide of carbon, arsenic, CO, and other poisons. These cases show a striking similarity to general paralytic cases, but differ from the latter in the suddenness of onset, the absence of grandiose delusions, and in the fact that they generally recover. This description of such cases under pseudo-general-paralysis has been recently assailed by many writers both in this country and in France, and ought to be abandoned. A number of these cases come under Korsakoff's psychosis, and the mental disorders may appear as the result of a toxæmia, without any appearance of paralysis or other lesions of the nerves. Musso's cases will be referred to in detail in the chapter on chronic poisoning.

Hysteria.—A number of cases of so-called hysteria following CO poisoning have been reported from time to time by French authors, some of whom go the length of affirming that all cases in which nerve symptoms are prominent are due to hysteria. Boulloche, [2] for example, held that all the cases of hemiplegia brought about by CO poisoning which occurred in young people were cases of hysteria. He quoted one case in which

[1] Musso: "Sulla pseudo-paralisi-generali per intossicazione lenta da ossido di carbonico," *Rivista clinica di Bologna*, 1885.

[2] Boulloche: *Archiv. de. Neurolog.*, 1890, p. 212.

blindness, asphasia, and deafness, lasting 24 hours, were followed by other nervous symptoms, all of which he put down to hysteria. A similar case is recorded by Laroche. The coma which ushers them in he regarded as a variety of hysterical apoplexy. Duponchel believed that a case of hemiplegia which came under his notice, and in which subcutaneous emphysema, visual disorders, and other nervous disturbances were exhibited, was also of hysterical origin.

Most of the cases referred to by these authors as hysterical cannot possibly in the light of modern knowledge be regarded as such. But there can be no doubt that CO is able to produce hysteria in people who are predisposed thereto, and some cases are on record where there could be little or no actual difficulty in diagnosis. Itzigsohn, [1] for example, reported a typical case of neurosis following CO poisoning. Of a number of girls who were exposed to CO gas, three became seriously ill and were rendered unconscious. Two recovered without any serious after-effects developing, but the third took an epileptiform convulsion, turning up her eyeballs and twitching her legs and arms. After a few hours she was again quite right. A week later, on the same day (Thursday) and at the same hour, she exhibited another fit. She recovered much more quickly from this attack. These attacks recurred on the next two succeeding Thursdays, and after that on four consecutive Wednesdays, at the same hour and in the same way. Under treatment she made a perfect recovery.

The following interesting case of hysteria, following exposure to illuminant-gas, was reported by Behr.[2] A man, 36 years old, was poisoned by gas from an escape in a pipe, and was found lying on the ground struggling violently. After some minutes he was able to rise, went towards his house, lay down on the stairs, and began to sob. A doctor pronounced his condition grave and sent him into hospital. On arriving there he gave confused answers to the doctor. At the end of some days he rose up and asked to go out. His voice was always tearful, and he had frequent attacks of weeping. Once he declared he was the victim of a plot to kill him, and that one of his subordinates was trying to make him lose his work. His father died an alcoholic, and a brother was weak-minded. One of his companions said he was always discontented and quarrelsome, while his vagaries and his arrogant character made him many enemies. At times he was inattentive and forgetful, suffered much from headaches, was often indisposed, but was regarded as an industrious and sober workman. He had a frightened and hesitating appearance. The pupils reacted to light, but unequally. Speech was normal, and there was no paralysis. He felt quite different

[1] Itzigsohn: *Virchow's Archiv.*, Bd. XIV., 1858.
[2] Behr; *Weiner. Med. Woch.* 1896, pp. 1716, 1768, and 1815.

from his usual; as he put it, he recognised himself with difficulty, he was a stranger to himself. He had beatings of the heart, and wished to rest the whole day without doing anything. He complained of certain peculiar sensations in his eyes, and of hearing bells before going to sleep. He would laugh frequently without knowing why, and often wept when he recalled his accident. His wife declared that he had completely changed; he forgot everything, repeated the same thing over and over again, and was constantly tired. His mood was constantly changing, he was often indifferent, sometimes morose, sometimes cheery and affable, at other times quarrelsome. Six weeks after the accident he complained of disturbances in the ears, notably of hyperacousis, of a sensation of continual rocking of the head, and of constantly shifting pains in the body. His attacks of weeping became less frequent. A week later he still complained of noises in the ears, and the pains were worse. A fortnight later he was sent to the country still complaining of the auditory disturbances. Four months after the accident he went back to his work. Six months after, while walking, he felt a severe pain about the heart, with a feeling of compression. This sensation disappeared very quickly at night, but came back during the day. He distinguished slight crises which lasted several seconds, and severe attacks which were more or less prolonged. As these crises became more frequent, his employer became alarmed and refused to employ him. After meeting his employers the attack appeared immediately; he spoke in a high tone, gesticulated, and wept. He also complained greatly of pain and constriction about the chest, of a ball in the throat, of tinglings, and of weakness.

Charcot[1] also showed the possibility of a latent hysteria awakening under the influence of an acute intoxication, and quoted the following unusual case where he considered *Astasia Abasia* (à forme trepidante) to have resulted from the effects of CO on a man who had a strongly-marked hereditary taint, and who had in a marked degree the hysterical diathesis. As this is the only case of the kind recorded, we give it in detail. The man's father was an alcoholic, and the members of the family were insane or ataxic. Three years before the accident he suffered from melancholia, which profoundly affected him both physically and morally. From that time he had attacks of suffocation and of indefinable weakness, which came on almost daily. Once he had an epileptiform seizure, which was preceded by an aura and accompanied by loss of consciousness. He was in this peculiar nervous condition when he was poisoned by CO, during convalescence from which the symptoms of abasia appeared. He lit a charcoal stove in the small room which he occupied and was poisoned

[1] Charcot: "Abasie à forme trepidante à la suite de l'intoxication par l'oxyde de carbone." *Leçon du Mardi*, 10th April, 1889, p. 355.

by the fumes. It was only after three days that he woke up mentally clear. On the second day he had spoken to a child who had come to see him, but he had no recollection of it. This amnesia, however, did not last long. He stayed in the Hôtel Dieu for twelve days, and just before leaving he began to experience some difficulty in walking. He was always oppressed by vague fears and terror of something portending. When lying in bed the patient was quite normal, as is usual in such cases; he could move his legs quite correctly and the muscular power appeared good, but once on his legs he experienced the greatest difficulty in walking. When he began to walk, the movements of flexion were counter-balanced by those of extension, and as Charcot describes it, he moved like a spasmodic paraplegic. The patient had a feeling that he would tumble backwards. There was no disturbance of sensation.

CHAPTER XII.

CHRONIC POISONING BY CARBON MONOXIDE.

How Produced.

The widespread and enormous use of gas nowadays for industrial purposes makes it very important that medical practitioners should understand and appreciate how gas-poisoning accidents arise, and how they may be recognised. Chronic poisoning by CO may be produced in many ways. Some of these have already been discussed at some length; for example, poisoning in connection with gas-engines, and more especially suction-gas engines; in blast-furnaces where the "bell" at the furnace-top is defective; in factories where gas is used for power purposes and has leaked into the workroom; and in cases where gas is used for fuel, with the consequent opportunities of leakage. We have also described a case of neuritis in a boy who was engaged in carrying shovels of blasting charcoal from furnace to engine fires (p. 236). Chronic poisoning also occurs among laundry-women, more especially the ironers, and among cooks who are exposed during their work to CO. The gas may be given off from charcoal stoves or by leakages from gas-cookers in badly-ventilated kitchens, and in laundries where gas and charcoal are used for heating the irons and for drying purposes. Lancereaux, Levy, and others, in reporting serious cases of chronic CO poisoning, held that such occurrences in cooks and ironers, who were exposed during their work to CO, were far from rare. It has been demonstrated again and again, where Bunsen burners are used for cooking stoves, that with great pressure gas may escape through the air-inlets in the Bunsen.

Albert Levy and Pecoul have carried out numerous and elaborate analyses of atmospheres which might possibly be contaminated by CO, and they have found that of the lighting and heating apparatus now in use some give rise to insidious gas-poisoning. It is held by some observers that, in not a few cases, ill-health among the working classes may perhaps be due to the use of "penny-in-the-slot" meters, and of gas cooking-stoves which are not connected with a flue. At the International Congress on Tuberculosis at Paris in 1905, Landouzy[1] declared that faulty construction of houses had a good deal to do with causing many

[1] Landouzy: *Lancet.* 11th November, 1905.

of the symptoms complained of by people living in apparently healthy houses. These symptoms, indigestion, headache, giddiness, palpitation, nausea, weakness, etc., which he regarded as the result of more or less continuous inhalation of small amounts of CO, were ordinarily put down by the doctor to overwork, functional or organic disease of the digestive organs, neurosis, improper diet, etc. In such cases, all treatment failed to cure the patient till they were sent to the country. Landouzy hoped that an active campaign would be instituted against the use of slow combustion stoves and leaking flues and chimneys, which caused such a high mortality, the effects of which, in his opinion, were undoubtedly one of the predisposing causes of consumption.

In France, especially in Paris, where slow combustion stoves are so popular, many deaths occur yearly as the result of acute poisoning by the fumes therefrom. Many practitioners of medicine have drawn attention to this fact, and there is a very full and complete discussion regarding the matter recorded in the *Bulletin de l'Academie de Médicine* of 1889. In these papers, several writers have drawn attention to the dangers of chronic poisoning arising from these stoves, owing to the toxic gases escaping therefrom into the atmosphere as the result of faulty construction, or of some defect in the method of carrying away the noxious fumes. Attention was also directed to the difficulty in diagnosing accurately the causes of such illnesses, as the ill-health which developed was so often put down to some other cause. Vallin,[1] for example, mentions that in two families who constantly used slow combustion stoves, some of the young members suffered from persistent headaches and pronounced anæmia. The illness, which in one case was put down to overwork at school, and in another to the stress of modern life, disappeared in both cases as if by magic with the return of good weather, which permitted an open-air life. Pettenkofer collected a number of cases of illuminant-gas poisoning which were regarded as typhoid fever. In one case the nurse and another person, who were looking after the patient, were affected; all were saved, the patient in an almost moribund condition, by being removed.

Similar cases have been reported in this country; one, for example, in Edinburgh, where the symptoms were regarded as due to typhoid fever, but were ultimately recognised as those of illuminant-gas poisoning. Many serious illnesses, regarded as pernicious anæmia or as serious organic diseases of the heart or stomach, have also been recorded, where it was afterwards discovered that they were due to illuminant gas and charcoal poisoning. In small, badly-ventilated rooms poisoning cases may easily arise from gas escaping from defective gas brackets and gasaliers. Workers about gas-works are also prone to suffer from CO poisoning. Cases of chronic poisoning by CO may arise, then, in those who work for

[1] Vallin: *Bull. de l'Academie de Médicine*, 1889, p. 425.

lengthened periods at a time in badly-ventilated rooms heated by defective, leaky, slow combustion stoves; in places where coke is used in open fire-places with defective chimneys; where gas stoves are used for cooking and heating purposes and the fittings are defective, more especially if there is a large proportion of water-gas in the gas burned; indeed, in all places where CO is given off, but in too small amounts to cause acute poisoning. It must not be forgotten that CO poisoning, owing to its cumulative action, may occur even in the open air, and cases of chronic poisoning may occur amongst those cleaning and repairing tubes about furnaces and ammonia plants, etc. We had a case of neuritis in a man engaged in such work.

The following interesting case, occurring in a linotype operator, was recently reported by a Factory Inspector. The printing office was a modern one and contained fifty-eight linotype machines. Each metal pot used in the process has a hood connected with a trunk and powerful fan-exhaust. A few months before the illness occurred, a gas plant had been installed to heat the metal pots. Owing to alterations in the position of the machines, which necessitated the hoods being disconnected, the gas which leaked out was not carried away at once. The Factory Department, in notifying the accident, added the following observations:—"Linotype and monotype machines need efficient exhaust-ventilation in order to minimise the danger from (*a*) fumes from the molten metal as well as (*b*) fumes from the burners below the pots, and also (*c*) to keep down the temperature."

Another case, also from the Factory Reports, shows how these cases may occur in gas-engine houses. A "clearance" or "sweeper-out" pipe had been left to end just outside the engine house, about 8 feet from the floor, near one of the producers which the man had daily to feed with fuel. The man was gradually poisoned by periodic escapes of gas into the room, although he was never rendered unconscious and never suffered from signs of acute poisoning. The room, it may be added, "was open to the air in parts."

Symptoms.

The points which have already been discussed at length regarding the establishment of toleration to small doses of CO must be remembered when dealing with chronic poisoning. Men who start work in gas-engine rooms, where there is always a small percentage of CO in the atmosphere, and the presence of which may be demonstrated in their blood, very often complain for weeks of various symptoms till toleration is effected, when they appeared to become well again. Even in such men, however, when a larger percentage of gas than usual is escaping, as, for example, when the engine has too little to do, these troublesome symptoms return. In other men we find that toleration never becomes established, and

when the symptoms become worse the men are forced to give up their work. Such cases are very much more common than is generally recognised.

The following are the symptoms generally complained of in such cases. One of the earliest is frequent and often intolerable headache, which, when very severe, is accompanied by nausea and vomiting. The peculiar feature of this headache in certain cases is that it does not come on till the patient gets out from work into the open air. In other cases it is most complained of when the patient wakens in the morning, just as is found in cases of alcoholic poisoning, although in a considerable number of cases it is present during the whole time the patient is at work in the poisonous atmosphere, and becomes more marked towards the end of the day's work.

Attacks of giddiness are very common. Sée, in 1873, remarking on the frequency with which this symptom was met with in persons suffering from chronic CO poisoning, referred to it as "le vertige des cuisines." The patient has not the usual control over the limbs, so that he staggers when he walks from muscular inco-ordination. Muscular weakness, which is also generally met with, accentuates this trouble in the lower limbs, and the fatigue of walking makes the patient stagger. He is very easily tired, and generally falls asleep as soon as he has taken his evening meal.

Derangements of the digestive system are common. Patients may have attacks of vomiting and nausea, and more rarely of diarrhœa. According to Hirt,[1] all gas-workers suffer from affections of the stomach, in his opinion produced by the constant inhalation of small amounts of illuminant-gas.

The cardiac symptoms, which have been already dealt with in detail, (p. 204) are very important and serious. Dr Fotheringham, who has had considerable experience of gas poisoning in large steel works where gas is very extensively used, has seen strong young men rendered physical wrecks by the constant inhalation of producer-gas, and in most of these cases the chief damage was on the heart. In some cases, cardiac dilatation may be made out, but in many of them no definite lesion can be discovered, the poison appearing to act chiefly on the nervous mechanism of the heart, probably on the vagus nerve. Palpitation, præcordial distress, and oppression about the chest, are very common symptoms in chronic CO poisoning. The pulse is often irregular, in some cases slowed, in others increased. The slightest exertion in some cases sends the pulse-rate upwards, out of all proportion to the amount of the effort. Another important symptom in some cases of chronic poisoning is, marked pulsation in the vessels of the neck. This may be present without any cardiac signs and symptoms.

[1] Hirt, quoted by Jaksch : *Die Vergiftungen*, 1910, p. 267.

Doreau[1] describes a case in which both cardiac and respiratory actions were found slowed. The pulse beat was 56 per minute, weak and compressible, and the respiratory movements, which were shallow and weak, were also slow and frequently interrupted by sighs. The patient complained of violent and continual pain in the chest, accompanied by a feeling of constriction and severe lumbago. She developed unaccustomed lassitude, showed no desire to move about, and complained of general weakness and of feeling ill. There were attacks of epigastric pain, accompanied by nausea and vomiting. Vertigo was a marked symptom, and also noises in the ears. The senses of smell, taste, sight, and hearing were all decreased, and tactile sensibility was considerably diminished, for the patient could hardly feel severe pinching or deep pricking with a needle.

The serious effect which CO has on the heart is well exemplified in three cases reported by Koren.[2] Three sisters, aged 4, 8, and 12 years respectively, were poisoned by CO from a slow combustion stove. The air of the room was found to contain 0·4 per cent. of CO, showing how the system becomes habituated even to what is commonly regarded as a fatal dose of CO. In these cases there was slight jaundice with attacks of vomiting, general pallor of the skin, headache, breathlessness, and dilatation of the veins of the neck. There were hæmic murmurs of the heart. The spleen was enlarged, as is sometimes noted in cases of anæmia, and there was dilatation of the heart. The number of the red blood corpuscles and the percentage of hæmoglobin were much diminished. Peptonuria was present. The eight-year-old girl died on the ninth day, and at the necropsy marked cardiac dilatation and advanced degeneration of the cardiac muscle were found. The others made rapid and complete recoveries. Commonly there is a feeling of illness and general weakness. At the same time the patient is not so alert as usual, nor is his memory so good.

The condition of the blood in chronic CO poisoning: Anæmia.

A very important point, which will require to be worked out much more carefully and in a more scientific manner than has hitherto been done, is the condition of the blood following chronic exposure to CO. We are of opinion that writers on both acute and chronic CO poisoning have been influenced too much by Claud Bernard's views of the physiological action of CO on the red blood corpuscles, viz.—that these corpuscles being in a manner destroyed, there was a great reduction in their number and in the amount of hæmoglobin, and that anæmia developed. Suc-

[1] Doreau: *Thèse de Paris*, 1881.

[2] Koren, quoted by Kobert: *Lehrbuch der Intoxikationen*, 1902.

ceeding writers took these changes too much for granted, and most of them contented themselves with noting the presence of anæmia, without giving any detailed description of the blood.

Anæmia, whether directly produced or due secondarily to changes in other organs, is certainly met with frequently in chronic CO poisoning, and is constantly referred to by most writers on the subject. Kobert[1] says that one of the principal signs of chronic CO poisoning is well-marked anæmia, of long duration without chlorosis and without hæmorrhages; and in all Musso's cases there was marked anæmia. In the three cases described by Koren[2], the number of red blood corpuscules and the percentage of hæmoglobin were diminished (in one case there being less than one million red blood corpuscles and in another 1,700,000), the spleen was enlarged, and the patients complained of other symptoms of advanced anæmia. Glaister,[3] in his evidence before the Departmental Committee on Compensation for Industrial Diseases, said "I have noticed fresh, healthy young girls go into a laundry, and within six months become absolutely bleached, colourless, anæmic, useless, and get knocked off work, and unless they were sent away to the country were apt to break down in health." Sachs[4] also saw cases of pronounced anæmia in laundresses in whom there were also digestive derangements, palpitation of the heart, and headache. These symptoms quickly disappeared when the operatives left their work, but reappeared when they resumed their occupation. He says that some cases are seen which show a striking resemblance to ordinary chlorosis, and some others so severe in type as to suggest pernicious anæmia. He quoted three cases by Torup,[5] where three children, poisoned by emanations from a portable stove, showed the symptoms and signs of pernicious anæmia.

In some of the cases of chronic poisoning which we have seen, there certainly was slight anæmia, but this we put down to the general deterioration of health. In one case, however, of a young man who had been working two or three weeks in a gas-engine house, whose blood we carefully examined, the character of the red blood corpuscles was not unlike that found in pernicious anæmia. The red blood corpuscles were irregular in shape and of different sizes, and normoblasts were present. The corpuscles, however, were much more numerous (5,600,000 per c.m.) and the percentage of hæmoglobin greater (92 per cent.) than usual. In connection with the subject of anæmia, it is

[1] Kobert: *Lehrbuch der Intoxikationen.* Stuttgart, 1902, p. 877.

[2] Koren: "Three cases in the same family of acute pernicious anæmia following chronic poisoning by CO." *Norsk. Mag.*, July 1891.

[3] Glaister: *Report of the Departmental Committee on Industrial Diseases*, 1907, p. 142.

[4] Sachs: *Kohlenoxydvergiftung.* Braunschweig, 1900.

[5] Torup: *N. Mag. f Laeger*, 1891. Quoted by Sachs.

interesting to remember that since Nicloux and St Martin pointed out that CO was present in normal blood and produced by the organism, Lepine and Boulud[1] showed that an increased percentage was present in the blood in certain diseased conditions, more especially in cancer and pernicious anæmia. They asked if the presence of this gas might not have a toxic effect and lead directly to the anæmia.

Certain observers have found a distinct hyperglobulia after both acute and chronic CO poisoning. Von Jaksch,[2] for example, found in the blood of a man who was recovering from serious CO poisoning, 6,390,000 erythrocytes and 13,100 leucocytes, and in two women who suffered from acute poisoning, 5,130,000 erythrocytes and 10,600 leucocytes, and 4,820,000 erythrocytes and 19,400 leucocytes respectively. In these cases the number of red-corpuscles gradually decreased, and Jaksch formed the opinion that this transitory increase stood in some relation to the CO poisoning. In all these cases the hæmoglobin was normal in amount; but in a case of acute poisoning reported by Sanger Brown,[3] although 5,230,000 erythrocytes were found, there was only 80 per cent. of hæmoglobin.

In their interesting experimental work, Nasmith and Graham[4] exposed guinea pigs for months to an atmosphere containing sufficient CO to throw out of action twenty-five per cent. of the blood corpuscles. They were surprised to find that, instead of loss of weight and anæmia resulting, the animals were as lively as before, and actually put on weight. At first there was degeneration of the corpuscles, but as early as the third day normoblasts appeared, and afterwards there was a steady increase in the number of red blood corpuscles, till in three weeks the maximum was reached. They concluded that just as is found in animals exposed to very high altitudes, the great increase in the number of red corpuscles in this case was Nature's defence or the result of compensation, the blood-forming organs working with greater physiological activity. When other animals were exposed to sufficient CO to cause 45 per cent. of saturation, although for the first few days there was great degeneration of corpuscles and the animals looked ill, soon an enormous increase in their number was found, the blood becoming quite thick.

The changes which go on in the blood of animals living in high altitudes are, as just remarked, not unlike the changes described by Nasmith and Graham. Many years ago Paul Bert[5] pointed out that

[1] Lepine and Boulud: Sur l'existence d'oxyde de carbone dans le sang des anémiques," *Compt. Rend. de la Soc. de Biol.* 1905. Vol. II.; and 1906, Vol. I., p. 302.

[2] Jaksch: *Die Vergiftungen*, 1910.

[3] Sanger Brown: *Journal American Med. Assoc.*, Chicago, 28th April 1906.

[4] Nasmith and Graham: *The Journal of Physiology*, 29th December 1906.

[5] Paul Bert: *Compt. rend. de l'Acad. des Sc.*, 1882, Vol. XCIV., p. 805.

animals, living in Mexico at a height of 1250 feet above sea-level, had twice as many red blood corpuscles as the same animals living at the sea-level. He found also that the blood of animals sent from a place in Bolivia which was 12,140 feet above sea-level, when shaken up with oxygen, absorbed considerably more than the blood of the same animals in Paris. This was the result of acclimatisation; but in the experiments carried out by Marcet,[1] this ability to absorb more oxygen took place a few hours after exposure to the rarefied atmosphere, and so could not apparently be due to any radical change in the constitution of the blood. In 1889, Viault[2] at Lima pointed out that in human beings the number of red corpuscles was considerably increased by residence in high altitudes, and this has been corroborated by many writers since then. Muntz[3] has contributed valuable work on this subject. He found that when animals lived at a great altitude where the oxygen tension was notably less, their blood became enriched in hæmoglobin, which would give a much greater oxygen-absorbing power in order to compensate for the rarefaction of the air. Koppe[4] proved that in rarefied air it was difficult for the corpuscles to take up sufficient oxygen, and he was able to demonstrate that within a few hours of reaching a place 700 metres higher up, the number of red corpuscles increased, and that many poikilocytes and microcytes appeared in the blood. He regarded these changes as an attempt by the organism to get a larger surface for the same amount of hæmoglobin.

The following cases by Reinhold[5] are interesting from the fact that they are the only cases of this type which have been described following chronic CO poisoning. In conjunction with the experimental work of Nasmith and Graham, and the nature of the changes found in animals and man at high altitudes, these cases throw new light on this subject.

Case 1.—A man, 40 years of age, was for many years a stoker in a gas-work. In 1902, two new boilers, which were fed by coke, were installed. There was always a smell of gas in the place. Owing to the collection of dust the flues and pipes had frequently to be cleaned. About nine months after the new boilers had been installed, the patient began to complain of always feeling tired, of frequent attacks of pain on the left side of the head, and of gradual loss of strength. Pains developed in the abdomen, and he lost his appetite. He also complained of prickling sensations along the inner side of the leg below the knee. On the 29th November 1902, he was forced to go to bed, where he remained till

[1] Marcet: *Contribution to History of Respiration of Man*, London, 1897.

[2] Viault: *Compt. rend. de l'Acad. des Sc.*, Bd. CXII., p. 295.

[3] Muntz: *Compt. rend. de l'Acad. des Sc.*, 1891, t. CXII., p. 298.

[4] Koppe, quoted by Reinhold.

[5] Reinhold: "Ueber schwere Anämie mit Hyperglobulie als Folgezustand chronischer Kohlenoxydvergiftung," *Muenchener Med. Woch.*, 26th April, 1904, p. 739.

March 1903. For a long time he could only move about with difficulty. On the 17th July 1903, he was admitted to the hospital. His weight was then 8 st. 4 lb., his skin was pale, the muscles flaccid, and all movements very feeble. On the head, face, and spine, parts were discovered which were insensitive to pressure and pain; and when the left leg was raised, considerable pain about the knee was produced; indeed, he had almost constant pain in the whole leg from the hip downwards. He walked in a very gingerly and cautious manner. The tendon reflexes were increased. There was no enlargement of the spleen. The pulse was 66, which increased to 84 on the slightest movement. The patient also exhibited alimentary glycosuria. The blood was examined with the following results:—

7th July.	8,225,000 r.b.c.	12,800 w.b.c.	Hæmoglobin	62 per cent.
8th July.	11,200,000 „	14,200 „	„	90 per cent.
9th July.	9,500,000 „	11,500 „	„	76 per cent.

At Reinhold's suggestion the Insurance Company sent the patient to a sanatorium at an altitude of 400 metres, where he remained three months. He increased eleven pounds in weight, but showed no other signs of improvement. The hyperglobulia was still marked, alimentary glycosuria could still be demonstrated, but there was a distinct increase in the hæmoglobin percentage shown as follows:—

5th Dec.	9,400,000 r.b.c.	9,800 w.b.c.	Hæmoglobin	100 per cent.
6th Dec.	11,000,000 „	14,700 „	„	90 per cent.
7th Dec.	10,200,000 „	12,400 „	„	90 per cent.

Notwithstanding the great increase in the number of red blood corpuscles, there was, for months, a distinct decrease in the amount of hæmoglobin, which lay between 62 and 90 per cent., the result of which was that each corpuscle had very little hæmoglobin. This would explain the extreme paleness of the skin, and also the anorexia and other symptoms due to want of oxygen in the tissues. Probably the great increase in the number of red corpuscles was brought about by a primary decrease in the amount of hæmoglobin caused by the action of the CO. In order to get a larger surface of hæmoglobin, the blood-forming organs would work with greater activity and produce the striking increase of red corpuscles which had been found.

Case 2.—The second case was that of a fellow workman of the previous patient. He was 45 years old, and had always been healthy till a year before he was examined, which was on the 15th January 1904, when he complained of stomach derangements, frequent headaches, and weakness. He was rather pale. Nothing abnormal was found in the heart and lungs, and the reflexes were normal. The blood was examined, and ound as follows:—

7,500,000 r.b.c. 14,000 w.b.c. Hæmoglobin, 100 per cent

Reinhold was of the view that the hyperglobulia in these cases was the result of permanent injury to the blood-forming organs, and that it was caused by the organism attempting to compensate for the disturbance by an over-production of the cell elements of the blood.

An interesting account of numerous cases of chronic poisoning was given by Morel and Mouriquand [1] in 1911. Of 40 persons who worked in a laboratory in Lyons, 35 presented very similar symptoms of illness, the cause of which was at first unknown. Later, it was discovered that these symptoms were due to CO poisoning, as the presence of CO was demonstrated in the atmosphere of the laboratory to the extent of from 1 part in 10,000 to 1 part in 1000. Numerous leakages of illuminating-gas which contained 12 to 14 per cent. of carbon monoxide were discovered, and this amount was increased during the winter months by escapes from defective joints of a heating apparatus. Nearly all these patients complained of various digestive derangements, loss of appetite, hyperchlorhydria, etc., and of various nervous disturbances, such as loss of memory, while examination of the urine revealed transient albuminuria and glycosuria. In all cases the symptoms disappeared during holidays, and were exaggerated during the winter months when the heating apparatus was used. Borczyskowsky [2] was the first to call attention to the appearance in the urine of sugar and albumen in chronic CO poisoning. Fraenkel, Araki, Munzer and Palma and others regarded the appearance of albumen in the urine as the result of the breaking down of the albuminous elements of the body, owing to the prolonged oxygen-starvation. This led to the circulation in the blood of toxins, the presence of which would also help to explain the numerous nervous and other symptoms so frequently encountered in both acute and chronic CO poisoning.

Chronic as well as acute poisoning may be followed by pneumonia. Lelorraine [3] described a typical case of chronic poisoning which was followed by an attack of congestion of the lungs. A woman of 28, who was employed in an underground cellar, lighted by gas and badly ventilated, complained of violent and continuous headache with a sensation of constriction in the temples, of difficulty in keeping awake, and of vertigo. There were also vague pains and weakness in the limbs, pain in the loins, and frequent attacks of palpitation with a feeling of oppression in the chest, which was more marked at night. Digestive derangements were also present, with rapid and marked loss of flesh. The patient frequently had attacks of vomiting in the morning. She developed a sharp attack of congestion of the lungs.

[1] Morel and Mouriquand ; "Chronic CO Poisoning." *The Lancet*, 1911, I., p. 63.

[2] Borczyskowsky ; "Die Chronische Kohlenoxydgasvergiftung," *Inaug. Dissert.* Greifswald, 1877.

[3] Lelorraine : *De l'Oxyde de Carbone au Point de Vue Hygiènique et Toxicologique.* Strasbourg, 1868.

Cerebral disorders are so frequently found after chronic CO poisoning that, as far back as 1869, Moreau de Tours,[1] father of the author of the first detailed study of mental disorders following chronic CO poisoning, was prompted to write as quoted by us already (p. 276). Before this, Chevalier,[2] in 1864, found certain derangements of vision. He examined a number of women exposed to constant inhalation of CO and found that they were all chlorotic, and that a considerable number suffered also from some disorder of vision, such as choroiditis, and in a few cases from optic neuritis. They could not use their eyes for any length of time without these becoming tired and painful. He found that recovery although slow, and in some cases requiring a year, was usually complete in all these cases.

Paralysis, etc.

The development of neuritis, paralysis, and other nervous disorders has already been fully dealt with, and cases following chronic poisoning have been described (p. 226). Such cases are not uncommon, and many more would doubtless be recorded, were medical men aware that such disorders may follow constant exposure to small percentage amounts of CO. The following is typical of such cases. A woman, aged 30, complained of constant headache, giddiness, faintness, and of impairment of the sense of touch, which not only prevented her guiding her needle, but also led her to believe that she was walking on something soft. Fibrillary tremor of the tongue, bad dreams, impairment of memory, digestive derangements, weakness of vision, and visual disorders as motes before the eyes, with discoloration of the skin, which was of a dirty muddy colour, were also present. The patient said that the symptoms began during the winter months three years previously when she had been using a stove; from that time she had suffered much from headache, giddiness, hacking cough, and irritable throat, and had, at frequent intervals, been compelled to stop her work. Levy,[3] who recorded this case, was of opinion that nervous disorders and other illnesses follow chronic poisoning by CO, and that these are much more common than reports would lead us to believe. There is good reason for believing that the rarity of these recorded cases is solely due to the fact that the symptoms are not well-known.

The Chief Factory Medical Inspector of the United Kingdom inquired into three cases of paralysis in men who had been engaged working on a gantry-staging feeding a furnace the bell of which, it was afterwards

[1] Moreau: *Des Troubles Intellectuels dus à l'intoxication lente par le Gaz Oxyde de Carbone*, Paris, 1876, p. 4.

[2] Chevalier: *Annales d'Hygiène Publique*, Vol. XXII., 1864, p. 48.

[3] Levy, quoted by Lancereaux, *Bull. de l'Acad. de Méd.*, 1889, Vol. XXI-XXII., p. 161.

found, did not fit properly and, therefore, allowed gas to escape. The workmen all complained of the same symptoms, viz.:—of giddiness, headache, and weakness of the legs, which gradually got worse. In each case typical steppage-gait was marked. In two, the grasp of the hand was very much weaker than normal. There was also slight paralysis of the right side of the face. Speech was affected in all three, in one very slightly, but in the third to such an extent that he could not say more than "yes" or "no," and could speak only in a drawling voice.

Molliet[1] was one of the first to call attention to the partial loss of memory which is frequently met with not only after acute but after chronic CO poisoning. One of his patients had the greatest difficulty in recalling the past, and always required a little time for consideration before replying to questions regarding her past history. The same observer also pointed out the frequency with which feebleness of vision, generally produced by congestion of the choroid, was met with in ironers in laundries, and he further noticed that in these cases this affection took a long time to disappear. The following case, described by him, gives a very good picture of chronic CO poisoning where certain mental derangements were prominent. A cook, 52 years of age, suffered from breathlessness on exertion, from violent attacks of oppression about the chest, and from palpitation, indigestion, and vomiting. Her appearance was that of a person with profound cachexia, the skin having the same dirty colour, while over the body, and especially over all the limbs, there was distinct œdema. The pulse was sixty-eight, and the heart-beat very feeble. When questioned, she replied slowly and with great difficulty. It was with considerable mental effort that she could recall antecedent events. No organic lesions could be detected. She recovered very slowly.

We have already called attention to the fact that cases of transient delirium after acute CO poisoning are comparatively frequent. This delirium is also occasionally met with in cases of chronic poisoning, and here it is generally prolonged, and very frequently accompanied by hallucinations of sight and hearing. To these hallucinations Moreau first called attention. He held that hallucinations of sight were not only common, but that they were invariable in chronic poisoning.

Poisoning was observed principally in women, and in the majority of the cases there was complete absence of all hereditary predisposition. It has been found, indeed, in recent times, that in works where CO poisoning cases arise owing to leakage of producer-gas into work-rooms, the women are the first to suffer, and that they are generally more seriously affected than men. Moreau also found that most of the women affected were over the age of 45. In these cases, however, we must take into con-

[1] Molliet: "L'Intoxication Chronique par l'Oxyde de Carbone," *Thèse de Paris*, 882.

sideration the usual instability of the nervous system at that period of life.

The following are the most characteristic symptoms which Moreau found in the cases which came under his own observation. In his monograph he describes twenty-seven cases. In nearly all, the patients complained of intense headache and compression of the temples, vertigo, dimness and disturbances of vision, flashes of light, of ringing and noises in the ears, of being easily tired, of general weakness, of attacks of faintness, and of hallucinations of hearing occasionally and of sight almost constantly. They also complained of a peculiar condition of mind characterised by indecision and wandering delirium, which he considered the most important phenomena of chronic poisoning. The last condition was often precipitated by a mental shock, such as a fright or accident, just as delirium tremens is often brought on by an accident. In some cases a very prominent mental feature is the mania of persecution, the patient developing a very suspicious frame of mind, constantly complaining that she is being pursued by people who wish to do her harm. Moreau says that these ideas are not very deeply rooted, for the patient can readily be persuaded, for a short time at least, that her fears are imaginary. A natural consequence of this mental condition is hallucinations of hearing; the patients hear men threatening and abusing them; there are hallucinations of sight, flashes of light, brilliant stars, showers of fire; and sometimes phantoms are very common. One of his patients had, in addition to ideas of persecution, hallucinations of sight of a mystic character, such as phantoms of all kinds, angels with whom she conversed, the animals of the Apocalypse moving about in the sky, etc. Patients may also see the phenomena peculiar to alcoholism:—animals such as rats, snakes, etc., but these are generally seen in the midst of flames, surrounded by flashes and sparks of fire. Hallucinations of smell and taste are very much less commonly noted. Moreau describes one case in which these were a prominent feature together with delusions of persecution.

The following is a short description of a few of the most interesting cases in Moreau's collection. A woman, 50 years of age, a cook, with no history of alcoholism, felt ill at the end of her day's work. She had attacks of fainting accompanied by buzzing noises in the ears, and of dimness of vision accompanied by flashes of light, sparks, and stars of fire. From time to time she complained of vague indefinite pains which always quickly disappeared in the open air. She was much agitated, very restless, and a prey to delirious ideas. She lived constantly in a state of dread, imagining at one time that she had been hypnotised, at another that someone was trying to poison her, at another that her body had been sold to the Medical School, and always that attempts were being made to do her harm. She heard her enemies speaking, but she did not know who

they were. She gradually improved, and although she still heard the voices of her persecutors, she was not so positive that they were not the creation of her own imagination, and sometimes she even laughed at the ideas she was told she had held. She made a quick recovery.

In another case, that of a woman 46 years of age, a mental condition developed quite the opposite of her usual frame of mind. From being a confident, self-reliant woman, she became timorous, indecisive, full of fears, and could no longer make up her mind. This state of mind did not last long, but it was succeeded by delusions She ceased going out of doors, as she believed that men, whom she did not know by sight, but whom she recognised by instinct as her persecutors, followed her to do her injury. She thought that attempts were also being made to poison her. She complained first to the police and then to the magistrate, with the consequence that her condition was inquired into. She also made a ɪapid recovery. In both of these cases there were no hereditary antecedents of mental weakness.

In another case, that of a woman aged 50, the husband declared that, for a long time before the acute mental condition supervened, his wife's mind had become changed. She had become irresolute, indecisive, and always hesitated and took a long time to make up her mind to do anything. Then she complained of hearing threatening voices, and of being followed in the street by people who meant to do her harm. At night she saw phantoms in luminous circles, she lived in an atmosphere of dread, and she had fears for the future and for the safety of her family, etc. This condition of imaginary fears and persecutions persisted so long that her husband was forced to have her confined. In another case there was marked weakening of the mental faculties and loss of memory. This woman thought that she also was the victim of plots, etc.

Abramovitsch[1] reported a case in which a woman, 44 years of age, after being exposed for a considerable time to small doses of CO, presented symptoms of mental disturbance in the form of melancholia, with hallucinations of the senses and ideas of persecution. She complained that people spoke ill of her, that her baby was threatened, and that for two months she had been pursued by a voice, which said all sorts of things to her and which prevented her sleeping. She also complained of painful disturbances of sensation,—pricking sensation all over the body, at one time of heat at another of cold, while occasionally she felt "as if she were pierced by electricity." She was afraid of the fire. She continually complained of bad odours in the room. Occasionally she became very excitable. When she was removed, for example, to the asylum, she made a great uproar, declaring that she had been bewitched, because she

[1] Abramovitsch: "Contribution à l'étude de l'Intoxication par l'Oxyde de Carbone," *Thèse de Lyon*, 1898.

had abundant menstrual flow which she declared had a very foul odour. Then she declared that somebody had given her itch, that vitriol had been thrown at her, that she had been burned, and that her whole body had been filled with strange and unpleasant odours. After an illness of three months she made a complete recovery.

Lancereaux[1] found cases of neuralgia, neuritis, etc., following chronic exposure to CO poisoning, in all of which certain mental disturbances, such as confusion of mind, weakening of the mental faculties, and partial loss of memory were present. The mental symptoms described may be present in all grades of severity up to actual dementia. Such cases have been described by various authors.

Musso[2] made a special study of these symptoms, and reported five cases, two of which recovered, the one after an illness of five months, and the other after nine months. In these the first symptoms complained of were frequent headaches, flushes of heat, giddiness, paresis, and disturbances of sensation of all kinds in the regions supplied by the trigeminal nerve and by a few spinal nerves. Some weeks afterwards other disturbances appeared, such as frequent attacks of palpitation of the heart and præcordial distress, sleeplessness, giddiness, mental dulness, formication in the legs, and a feeling of general weakness. Then appeared apoplectiform and epileptiform convulsions, followed by considerable weakening of the mental power. The muscles gradually became weaker, and disturbance of muscular co-ordination also occurred, so that all movements, including those of speech, became difficult. There were symptoms of advanced anæmia, and the patient lost flesh rapidly. The tendon reflexes gradually diminished till they almost disappeared; the pupils reacted sluggishly, and sometimes not at all; there were disorders of speech; and the whole picture reminded Musso of a commencing general paralysis. The epileptiform convulsions became worse, and cerebral hæmorrhages terminated the scene. Three of the five patients, after the symptoms of cerebral irritation disappeared, became imbecile—typical cases of *dementia paralytica.*

[1] Lancereaux: *Bull. de l'Acad. de Méd.*, 1889, p. 167.

[2] Musso: "Sulla pseudo-paralsi generale per intossicazione lenta da ossido di carbonio," *Rivista Clinica di Bologna.* 1885.

CHAPTER XIII.

TREATMENT.

With regard to treatment, the first thing to urge is that no case, however serious it may at first appear, should be regarded as hopeless. For example, the following case was quoted many years ago by Chevalier [1] where a man lay in a desperate condition for hours and yet recovered, in spite, it may be added, of the treatment adopted. The man was found apparently dead, and was taken outside into a courtyard and exposed to the cold of winter for half-an-hour. His face was swollen and of a bluish tint; he was deeply comatose; there was complete relaxation of the limbs; and there were no signs of any action of the heart and lungs. After a time warmth and friction to the skin were applied. For a long period every restorative measure seemed to fail, and death appeared imminent, when one of the doctors with his stethoscope thought he heard a slight sound in the lung. After three hours this became more apparent, and a mirror put to the mouth showed signs of breathing; then bleeding was tried, and after eleven hours' hard work, at length they succeeded in getting the heart and lungs to work more regularly, although the patient still remained in a deeply-comatose condition for many hours thereafter. He eventually recovered.

The greatest care is required in watching a patient, as relapses are very common, and one who is supposed to be out of danger may suddenly develop serious symptoms. In all cases the breathing should be carefully observed to detect any tendency to failure, and the same may be said of the heart. Indeed, every possible care is required if the patient is to escape serious after-consequences.

In carbon monoxide poisoning we are dealing with a gas which has entered into combination with the hæmoglobin of the red-blood corpuscles, a combination which is most difficult to disturb, and which robs the blood of a lesser or greater part of its oxygen-carrying capacity. What ought we to do? The patient should first be quickly removed from the poisonous atmosphere and carried into pure air. This is generally into the open air. But where this is done, the greatest care must be taken to prevent chilling of the body-surface, by providing that the patient is well wrapped up and kept warm by warm blankets, hot-water bottles, heated flasks, etc. As it is most important to prevent any further lower-

[1] Chevalier: *Annales d'Hygiène Publique et de Méd. Légale*, 1864.

ing of the body-heat, it is preferable, when possible, that the patient should be placed in a large airy room. Every large work and every colliery ought to have such a room available at all times for the treatment of accidents, and the room to contain appliances which are available and useful in first-aid for accidents. The second, is to provide a copious supply of fresh air or of oxygen, and by deep and rhythmic movement of the thoracic walls and respiratory muscles, to assist the lungs to take up an adequate quantity of good air or oxygen, which will, in course of time, slowly it may be, but surely, displace the CO from the hæmoglobin, and thus once more supply the tissues with oxygen.

Posture of the Patient.

Since the nervous system is most seriously affected by CO, and is also the tissue which exhibits most the deprivation of oxygen, it is very necessary to keep it well supplied with blood, or in other words with oxygen; consequently the best position for the patient is on the back with the head low and the limbs raised, just as one would treat a patient who has had a dangerous hæmorrhage. In less serious cases, keeping the patient at rest with the head low will often get rid of some of the distressing symptoms, such as vertigo and noises in the ears, while it usually relieves very considerably the headache and disturbances of vision.

Artificial Respiration.

Many years ago Hoppe-Seyler, who was one of the first to consider the action of CO scientifically, found when animals had been seriously poisoned by CO, that they were quickly restored by artificial respiration. He strongly advocated its use in all cases, as he considered that by this means the gas was oxidised into CO_2 in the blood. Thomas,[1] to whose pioneer work on mine gases we have already alluded, called attention to the value of artificial respiration in those who were overcome by after-damp in rescue operations after explosions. He thought this would be much better than the administration of alcohol, which was at that time the usual method of treatment. In the public press he called attention afterwards to the great value of oxygen in cases of poisoning by after-damp and the fumes from underground fires.

Artificial respiration should be persisted in till the breathing becomes sufficiently deep to supply as large an amount of fresh air to the CO-charged hæmoglobin as will enable the blood to give up its CO and have it replaced by oxygen. Where the respirations have been shallow, as, for example, where the patient has been deeply unconscious for some time, this is specially important. It must be remembered that the red

[1] Thomas: *A Treatise on Coal, Mine-Gases and Ventilation*, 1878. p. 185.

corpuscles, after association with the CO, take up oxygen readily as far as their hæmoglobin has not been mortgaged by this forced combination with the gas. Half an hour to one and a half hours generally suffices, even in bad cases, to get rid of a considerable quantity of CO, and thus to place the patient beyond immediate danger. It must not be forgotten, however, that the displacement of CO from the blood is slower than its absorption. Haldane believes that in serious cases, that is to say if the blood be saturated to about 70 per cent., it would take about six or seven hours for it to free itself entirely of the combination. It is important to insist on this fact, because many of the immediate after-effects and sequelæ following gassing are put down by miners and furnacemen to the poison being still in the system, and we have even heard medical men speak of its being in the blood for two or three weeks.

Administration of Oxygen.

In every work where cases of poisoning by CO may from time to time occur, cylinders of oxygen ought to be kept in readiness. This gas proves of the utmost value in such cases. Regarding its value in CO poisoning, a number of observers, such as Gréhant, Lesser, Haldane, Mosso, and others, have proved that animals may breathe with safety a very large percentage of CO in the presence of oxygen under pressure. Haldane [1] showed that CO was less poisonous the higher the tension of oxygen in the atmosphere, and if the tension were increased to two atmospheres, that very large percentages of CO could be breathed with impunity. In an atmosphere containing large percentages of CO and oxygen under high pressure, the red blood corpuscles even then combine readily with the CO, and the value of the high tension of oxygen in preventing the poisonous effect of the CO does not lie in the fact that it prevents the CO combining with the hæmoglobin.

Haldane's explanation is that, at a pressure of two atmospheres of oxygen, enough of this gas is dissolved in the plasma under high-tension to support respiration without the aid of the red corpuscles, the latter being completely thrown out of function by the CO; that is to say, the oxygen in solution is of the greatest value in saving the oxygen of the corpuscles, and where the absorption of oxygen is very great, that an animal can live without calling upon the oxygen of the hæmoglobin. Gréhant[2] found that when a dog breathed an atmosphere containing 1 per cent. of CO, it died in about twenty minutes, but in a mixture of oxygen and 1 per cent of CO it might live $2\frac{1}{4}$ hours or even longer. Mosso [3] confirmed Haldane's work. Experimenting with such animals

[1] Haldane: *Journal of Physiology*, Vol. XVIII., p. 295.

[2] Gréhant: *L'Oxyde de Carbone.* Paris 1903, p. 183.

[3] Mosso; *La Respirazione nelle gallerie e l'Azione dell ossido di Carbone.* Turin 1900; also *Compt. Rend. de l'Acad. des Sc.*, 1900, p. 483.

as dogs and monkeys, he found that a mixture containing 6 per cent. of CO showed no harmful results, provided that oxygen was present under a pressure of two atmospheres or air at a pressure of ten atmospheres, whereas on the other hand 0·5 per cent. of CO would canse death at ordinary pressures. He also found that if the animals were brought straight out of the mixture of oxygen and CO into the air they died at once, but that, if the mixture were gradually purified, "the blood was regularly washed," and that in about half an hour they could be placed in a normal atmosphere.

Much scientific work has recently been carried out in this country by Haldane, Lorraine Smith, Leonard Hill, Macleod and others, regarding the physiological effect of breathing oxygen, and the dangers of oxygen poisoning. The pressure at which oxygen is inhaled is a most important matter, and it has been found when the exposure is prolonged and continuous, the pressure being at the same time high, that inflammation of the lungs, convulsions, and other nervous conditions may result. Lorraine Smith also proved, where the tension of oxygen was very high, that the power of the lungs to absorb oxygen was diminished. In his experiments he found that, where more than 75 per cent. of oxygen, *i.e.* about four times the normal oxygen of the atmosphere, was administered continuously, pneumonia developed, but that 150 per cent. might be breathed with impunity for such short periods as one hour. In everyday practice, it is found that an atmosphere of oxygen can be breathed without serious result for two or three hours a day for a number of days. It is well to remember at the same time, however, that by the method by which oxygen is generally administered, a very large percentage of the gas escapes into the atmosphere. Leonard Hill[1] has pointed out that in the Hudson tunnel operations, mules were exposed for over a year to a pressure of air which gave a partial pressure of oxygen of 60 per cent. of an atmosphere, and that they remained in perfect health. He also quoted Bornstein's experiments on himself and on two others in the Elbe tunnel works, which proved that three atmospheres of oxygen (95 per cent.) could be breathed for half-an-hour with impunity. Haldane advises the use of a mixture containing 50 per cent. of oxygen, in order to do away with any risk of oxygen-poisoning.

Regarding the absorption of oxygen, the old idea was that the more the oxygen administered the greater was the metabolism in the tissues, but we now know that there is nothing to be gained by administering oxygen under considerable pressure. It has been proved by physiologists that, in giving one atmosphere of oxygen, metabolism in the tissues is not increased by the further increase of the gas, as a fire would

[1] Leonard Hill: *Recent Advances in Physiology*; also *The Colliery Guardian*, 17th March 1911.

be; that is to say, the tissues cannot utilise more than the normal amount of oxygen, and a further increase in the amount inhaled has very little effect on the capillary oxygen-tension. Indeed, Lorraine Smith [1] pointed out that high partial pressures of oxygen, which in time would produce in themselves poisonous effects, lead to lessened metabolism and to direct diminution of the body heat, an effect to be avoided in CO poisoning.

Still one other fact is of value in proving the enormous value of oxygen administration. Gréhant in his experiments showed, when oxygen was inhaled by persons suffering from CO poisoning, that it could drive out the CO five times as quickly as ordinary air. Haldane also verified this observation. From his experiments, Haldane concluded that, although there was not altogether conclusive evidence regarding the efficiency of oxygen, there could be no doubt that its inhalation hastened the elimination of CO, and also provided the tissues with an important supply of dissolved oxygen.

The following case by Lt. Col. Henry Elsdale[2] shows the value of the exhibition of oxygen in gas-poisoning cases. A soldier engaged in emptying a war-balloon was overpowered by the gas. When rescued he was to all appearance dead, his heart had ceased beating, and there was no perceptible breathing. After a little time, as there was still no sign of life, Col. Elsdale sent for a cylinder of the oxygen gas used for the oxy-hydrogen light, which contained pure oxygen at a pressure of over 1000lb. to the square inch. He forced open the sapper's mouth and inserted the nozzle of the valve inside, and gently turned on the valve. The oxygen under the great pressure must have entered all parts of his lungs in a very short time. "The effect was magical. He revived and clutched the nozzle with his teeth. For a few seconds the oxygen was pouring into his system at a very high and steady pressure." He developed convulsions, however, but soon recovered, and suffered from no ill after-effects. The administration of the oxygen under such high pressure in this case was happily short. Had it been prolonged, serious effects might have been produced.

In the chapter on rescue work in mines, we draw attention to the uses and value of the "Pulmotor," either as a means of administering oxygen or as a method of artificial respiration combined therewith.

Hill, who has done much with regard to rescue work in mines, has pointed out the futility of the usual method of administering oxygen. This, generally given through a nozzle held to the patient's mouth and nose, is quite ineffectual, as most of the oxygen escapes into the atmosphere, which thus contains but a very slight increase of the normal oxygen

[1] Lorraine Smith: *Journal of Physiology*, 1899.

[2] Elsdale: "Resuscitation by Oxygen," *Nineteenth Century*, May 1891.

percentage. He[1] advises the use of the following apparatus:—A celluloid face-piece, to which is attached a curtain of washable material, is attached to the cylinder of oxyen, the handle of the mask being the inlet tube for the oxygen. The current of oxygen, as it passes over the face of the patient should be forcible enough to produce a sensation of coolness which is very agreeable and comforting to the patient. In this way a very high percentage of oxygen, about 70 per cent., may be obtained in the alveolar air. He also thinks that the inhalation of alcohol vapour with oxygen, which has been demonstrated by Wilcox and Collingwood to have a beneficial action on the heart, might be used in cases of CO poisoning. A mixture of two parts of brandy or whisky with one part of water placed in a heating box is vaporised by an electric lamp placed in the bottom of the box through which the current of oxygen is led.

Oxygen may be used for other purposes as, for example, clearing the tainted atmosphere in places where men are working. The serious consequences of the accident reported (p. 295) in which a man, who was overcome in an exhauster in connection with an ammonia work, and could only be got out by the place being taken to pieces, would have been avoided if a cylinder of oxygen had been at hand and the poisonous gas thus driven out. Indiarubber bags could be used for transporting the oxygen from the steel cylinders to the scene of the accident.

At the Archduke Frederick's works in Austrian Silesia in December, 1896, six men were overcome by gas while working inside a boiler fired by furnace-gas. All the men were rendered unconscious. After several hours had elapsed, the engineer allowed 70 cubic feet of oxygen from two cylinders to flow into the boiler. In a very short time the breathing of the victims could be heard, and soon thereafter four were able to crawl out. The others, who lay between the sides of the boiler and the furnace tubes, were shortly afterwards brought round after two other cylinders had been used.

In large works where compressed air is used, as in the blast for the furnaces, for the automatic hammers, etc., in steel works, the air could be led into any place where accumulations of noxious gas were suspected, and the gas thus driven out. This would allow the men to work with impunity in many places where "gassing" cases are repeatedly found. In the mines in South Africa compressed air is used for ventilating sections where the air is very bad.

At this point it is interesting to remember a method which, until quite recently, was in common use in all cases of gassing in pits and in blast-furnaces both in Scotland and England, by which the gas was

[1] Hill: "The Administration of Oxygen," *Brit. Med. Journ.*, 13th January 1912.

supposed to be drawn out of the lungs. We have encountered a number of men who have undergone this treatment. It was as follows :—When a man was gassed (if it happened at the furnaces), he was carried at once to the fresh sand which is used for making the pig-beds, this sand being generally a little damp (sand which had already been in use on the pig-beds never being used for the purpose), a shallow grave was dug in this sand, a handkerchief placed over the man's face to keep the sand out of his eyes, etc., and he was covered completely, the sand being smoothed carefully all over his body. Any signs of rippling, creasing, or breaking of the sand was supposed to show that the sand had worked its cure, and the man was then lifted out of his temporary grave. The method was also used for resuscitating birds which are so frequently poisoned about furnaces.

A fatal case happened at Shotts ironworks, where a man was taken out quite dead after being so buried for some time. This, and other cases elsewhere, gradually shook the faith of the majority of workmen in the efficacy of this extraordinary line of treatment, although a number who still believe in it may yet be found. The men held that the sand in some way drew the gas out of the lungs. Why this unusual and barbarous method came to be used, and what good they thought it would do, or in what way it was supposed to act, we are unable to say. The older men, nevertheless, declare that this treatment has a wonderful effect in bringing an unconscious man round, especially where the breathing is very quiet.

We understand that the method adopted in collieries and at furnaces in certain parts of the country differs somewhat from the above. Dr Grant of Blantyre, who has an exceptional knowledge of miners, who saw all the survivors at the great Blantyre and Udston disasters, and who personally has seen the following method carried out in many cases, has told us that the method there adopted appeared to effect its purpose. A sod was cut out of the turf, and the man, who was brought up poisoned by after-damp and in an unconscious condition, was placed face downwards in this and his head covered.

The following is another form of treatment which was advised by the French Authorities about 1850 for poisoning by after-damp, etc. This is taken from a report by Mr Matthias Dunn, about the year 1852 :

(1) The person should be first removed into the open air, when he should be undressed and effusions of cold water thrown over his body; (2) Endeavours should be made to make him swallow cold water slightly acidulated with vinegar; (3) Clysters should be given, two thirds of cold water and one third of vinegar; (4) The pituitary membrane should be irritated with a feather, or stimulated with a bottle of volatile alkali put under the nose; (5) Air should be introduced into the lungs by

blowing with the nozzle of a bellows into one of the nostrils and compressing the other with the fingers, at the same time endeavours should be made to give motion to the chest, etc. It was therefore suggested to anticipate explosions by having prepared ready a supply of water mixed with a little brandy, and also a couple of common house-bellows, to be applied as soon as the person was removed into the fresh air.

Bleeding.

Bleeding has been mentioned as a remedy which was formerly widely employed, based, however, on erroneous views regarding the stability of hæmoglobin. Artigalas, for example, in his thesis on "Des Asphyxies Toxiques," writes in 1883 as follows :—"In CO poisoning abundant bleeding, having for its end the elimination from the vessels of corpuscles which are dead to their function, that is to say of CO-hæmoglobin which is incapable of becoming oxyhæmoglobin, is called for. This depletion of blood appeared to me necessary in order to disembarrass the vessels of the blood which causes obstructions there, and in order to permit the blood-forming organs to pour into the circulation the result of their functioning." From our modern knowledge, however, regarding the action of CO, we are of opinion that the routine exhibition of this treatment is most unscientific. By abstracting blood we are lessening the body-heat, while in no way improving the percentage relation of oxygen to CO in the remaining blood. While we condemn bleeding as a routine method of treatment, however, there can be no doubt whatever that in certain cases, for example where there is marked cyanosis and a dusky tinge of the skin, bleeding may do good by relieving the engorged circulation; and many cases are on record where it was only after venesection was performed that the patient commenced to rally. Glaister[1] notes the efficacy of bleeding in a case of CO poisoning even after failure from the use of oxygen. In other cases where the coma was very profound, it has also given good results. Kuhne, to whose work we shall have occasion to refer, in his experiments in transfusion, found where the condition was so serious that the heart was practically at a standstill and the respiratory rate one per minute, that the animal might recover if it were bled, whereas it always died where venesection was not carried out.

Transfusion.

Traube,[2] in 1864, was the first to employ transfusion in CO poisoning. He used it for a man who had been overcome by charcoal fumes. After the patient had been repeatedly bled, Traube injected about eight ounces

[1] Glaister: Op. cit., *The Lancet*, p. 357; Text-Book, p. 658.
[2] Traube: *Verhandl. d. Berlin, Med. Ges.*, Bd. I., 1864, p. 68.

of defibrinated blood into the cephalic vein. The patient died. Wagner about the same time also employed this method, but with an equally unsuccessful result, in a boy of thirteen who had been poisoned by CO. At the end of the same year, Kuhne[1] carried out a large number of experiments on animals to prove the value of this method. The transfusions were all made with dog's blood which had previously been carefully defibrinated and filtered. It was then aërated, heated to 35°C., and injected into the jugular vein. At the same time an equal quantity of the toxic blood was withdrawn. The following were the conclusions he arrived at:—(1) Animals rendered deeply insensible recover without artificial respiration being resorted to, provided the respirations have not dropped below two per minute. (2) If the respiratory rate, however, drops to one per minute, artificial respiration is necessary for recovery. (3) If the respiration is suppressed for some minutes, recovery is impossible, even if the heart is still beating, if bleeding and artificial respiration only are employed; but where transfusion of blood is used, recovery is possible, even when the respiration has ceased for seven minutes and where the beating of the heart has apparently ceased.

Not long afterwards, Halstead[2] advised the removal of a quantity of the vitiated blood, which, after defibrination and oxygenation, was restored to the circulation. This observer believed greatly in venesection, and maintained that bleeding ought to be persisted in even after the patient was out of danger. He thought that saline infusions might prove as useful as blood. After this date transfusion was employed occasionally, but it was only after the work of Leyden[3] that it was used by many as a routine method of treatment. In 1888, Leyden pointed out the great value of a rational exhibition of this treatment. He found in one grave case of poisoning, after all methods had failed and when the patient appeared almost lifeless, that an injection of 250 c.c. of blood taken from the body of another patient and injected into the veins, caused, after an interval of a few hours, signs of life to appear. Shortly afterwards the patient fell into a sleep lasting ten hours, and woke up well.

In 1889 an important contribution appeared on this subject by Laborde and Gréhant.[4] In it they discussed at length Kuhne's results. They held that transfusion was all the more necessary because death occurred in every case before all the hæmoglobin of the blood was saturated with CO; also that the injection of oxygenated blood, if it

[1] Kuhne: *Centralbl. f. d. Med. Wiss.*, 1864 p. 134.
[2] Halstead: *New York Med. Journal*, 8th December, 1883.
[3] Leyden: *Verhandl. d. Vereins f. innere Med.*, Berlin, 1888.
[4] Laborde and Gréhant: *Bull. de l'Acad. de Méd.*, 1889, p. 531.

did no more, gained precious time during which other methods could be carried out, and each hour gained increased the patient's chances of ultimate recovery. They believed that the most rational method suggested by a knowledge of the physiological mechanism of CO poisoning was transfusion of blood, and that this was confirmed by their experimental work. Their first experiments were directed to find out the extreme point of poisoning at which treatment might be successful. After most careful experimental work, they decided that the point had been reached when the respiratory capacity of the blood had fallen to one-half or even one-third of the normal. Up to this point bleeding and artificial respiration alone might succeed; that is to say, in cases where the respiratory rate was seven per minute and the cardiac beats eight to ten per minute. Regarding transfusion, they found that in order to be efficacious it ought to be carried out when the contractions of the heart and lungs, however feeble and slow they might be, had not completely stopped. Regarding the respective merits of bleeding, transfusion, and artificial respiration, they supported Kuhne's conclusions. They found that some seconds after transfusion definite signs of life were produced, the heart became reanimated, and the respiratory movements stimulated in an animal which, without this method, would have died.

They then carried out experiments to find out the value of simple bleeding, of bleeding associated with artificial respiration, of artificial respiration used alone, and of these combined with transfusion. Regarding artificial respiration, they found in cases where the breathing became very slow, and where the heart-beat was almost imperceptible, that artificial respiration, even if continued for a long time, was absolutely inefficacious. Other experiments were carried out to find out whether, after bleeding and artificial respiration had failed, transfusion would succeed. This was always successful, even in the gravest case. Bleeding and artificial respiration succeeded only at less advanced stages of poisoning. These methods, combined with transfusion, appeared to hasten sensibly the results of the latter, but they were not absolutely necessary. They concluded, therefore, that bleeding, followed by transfusion and combined with administration of oxygen, constituted the most powerful and efficacious method of treating CO poisoning.

One of the strongest advocates of bleeding combined with transfusion is Jaksch,[1] who, since he adopted this method of treatment, has not lost one of nineteen cases, some of which were very serious, and several in the last stages and apparently hopeless. He found that unconsciousness was strikingly shortened, and that all the patients recovered very quickly. He withdraws 300 to 400 c.c. of blood, and then gives a subcutaneous infusion of normal saline solution into the thigh. He also administers

[1] Jaksch: *Die Vergiftungen*, 1910, p. 265.

oxygen and, if the comatose condition continues, he advises that the patient should be put into a warm bath and cold water poured over the head. He has rarely, however, had to resort to the latter method.

Warmth.

In very exceptional cases it has been noted that, immediately before death from CO, the temperature reached a very high point (just as in fevers where there is a marked toxic action), owing to the heat-regulating centre being thrown out of gear, either on account of the direct action of CO, or of the greatly-lessened supply of oxygen due to the combination of CO with the hæmoglobin of the blood. But as we have already pointed out, one of the most serious results of CO poisoning is the lowering of the body-heat, either by diminished production of heat or by increased loss. In Haldane's experiments to determine the minimum poisonous dose of CO, he found that with small animals its action depended to some extent on temperature. A low temperature leads to increased metabolism with increased demand for oxygen, with the result that the animal is more easily affected by CO. In man, with decrease of external temperature, there is not the same increase of metabolism, but if the atmosphere is cold, it must have a serious effect on the body-heat.

No time should be lost, therefore, in increasing the body-heat of the patient by the ordinary means, both external and internal, usually at our disposal. Energetic friction of the skin in an upward direction is also most useful in increasing the body-heat and also in stimulating the circulation. A caution must be given against the indiscriminate exhibition of alcohol. In all accidents of this kind one generally finds that more or less raw whisky has been given to the patient. But alcohol only tends to reduce still further the body temperature. If a stimulant is to be used for obtaining warmth, it should be in the form of weak alcohol in hot water, or preferably of hot, strong coffee or tea.

The preservation and increase of the body-heat of the patient is not, perhaps, insisted upon so forcibly as it should be by some writers, and it is more than likely that lives have been lost, and recoveries retarded, owing to inattention to this most important point. The old teaching, indeed, was firstly and chiefly to give the patient plenty of fresh air, no matter how cold it was, plenty of whisky, an emetic, and, where necessary, to perform venesection. Dr Grant of Blantyre, when attending the survivors of the Blantyre and Udston disasters, was very much struck by the fact that men who were conscious and had apparently recovered, often became unconscious suddenly, and in some cases died, when they were brought up from the comparatively warm mine and were exposed to the cold atmosphere. This is a fact well-recognised in rescue work in mines, for it is often found that when the men reach the cool air at the

pit-bottom where there is a considerable current of air, they collapse. It must also be remembered that miners generally work in a half-naked condition. Haldane, in his experiments with small animals, found that they recovered much more quickly in fresh air when heat was applied. Regarding this fact of men collapsing when brought up from below, Mosso[1] in a recent article, advised for its prevention the placing of the patient in an atmosphere of compressed air or oxygen, as he considers the collapse is produced by alterations in the oxygen-pressure in the blood. In all collieries and iron and steel works in which gas-poisoning cases may occur, there should always be, therefore, a supply of blankets, etc., at hand. Haldane and several German authorities advise giving a warm bath to sufferers, as this is calculated to restore more quickly the body-temperature.

Rest.

As important organs may be seriously affected by the enforced deprivation of oxygen, rest is absolutely essential in cases of CO poisoning. When called to a case of gassing in works, one generally finds that the rescuers, if the patient has become conscious, begin to walk him about to keep him roused, and that it is quite common for the patient to collapse again, when artificial respiration has to be employed. When patients have been walked about too soon, they are usually longer in recovering. When a man has been rendered unconscious by gassing, complete rest should be insisted upon; he should make as little exertion as possible for at least six hours; and should on no account be allowed to walk home. Any exertion only uses up the small amount of oxygen left in his blood, with the result that he then becomes breathless, complains of giddiness and disturbances of vision, staggers about, and may even fall to the ground in an unconscious condition. The fact should never be lost sight of, that the longer the organism is deprived of a proper supply of oxygen, the more likely are serious after-effects to develop.

In mines, when men are working in an atmosphere where CO is present, it is generally found that it is the man who is working hardest, that is, "backing" out the coal, who is the first to complain and feel the bad effects. The great danger of muscular exertion in bad air was very well demonstrated in the Snaefell disaster. If the men had not hurried so much, several lives might have been saved. Consequently, rescue parties in such disasters should be warned to proceed very slowly, and to take the greatest care in going along difficult places, as the increased speed and greater exertion in climbing over falls mean increased respiration and a larger amount of CO inspired, with, at the same time, owing

[1] Mosso: "Action physiologique et applications thérapeutiques de l'oxygène comprimé," *Compt. Rend. de l'Acad. des Sc.*, 1900, p. 483.

to the work performed, more rapid metabolism and expenditure of the oxygen of his blood, which the person can not afford to lose.

Probably one of the best examples which can be given of the effect of exertion on a person suffering from CO poisoning is that furnished by the experimental work of Haldane[1] on himself. The degree of saturation of blood was 27 per cent. He describes his sensations as follows:—"Immediately after blood was taken (for examination) I ran twice up and down stairs. This caused for a minute or two distinct dimness of vision and hearing, and a slight tendency to stagger, besides abnormal hypernœa." With saturation at 37 per cent:—"I felt dull and abnormal. On running once upstairs I became very distinctly weak in the legs and had to lean against the table. At the same time vision and hearing became markedly affected, and there seemed to be some confusion of mind. The sound of the running water of the aspirator seemed to be far away, and to come much nearer after I had rested for a minute or two, vision clearing up correspondingly at the same time. While vision was impaired, I found it difficult to distinguish the tints of the different specimens of blood. Two or three minutes later I ran upstairs again with the same results. The symptoms came on about half a minute after I reached the top of the stair. On testing with a watch, hearing appeared distinctly impaired."

Mr D. M. Mowat,[2] while making experiments with a gas-scrubber, was poisoned by CO. He had to go down a ladder fully 20 feet long, and by the time he reached the foot he had lost his sight, although he was quite conscious. After three or four minutes he recovered, but received a message to go to the office. He walked rapidly uphill to the office and again his sight failed him, although he did not lose consciousness. He recovered once more after two or three minutes and afterwards walked slowly home, when he again felt the approach of the same symptoms, which, however, on this occasion did not cause loss of sight.

Emetics.

Emetics were formerly given, indeed were part of the routine treatment, and even yet it is not an uncommon thing to find that the workmen have administered a good dose of salt and water to the patient as soon as he has regained consciousness. This, it is believed by them, "clears the system of the gas" he is supposed to have swallowed. We have already alluded to the fact that certain patients complain of severe epigastric pain which is generally relieved by the vomiting of bilious matter, and that this, as well as the idea of ridding the system of the poison by sick-

[1] Haldane: "The Action of Carbonic Oxide on Man," *Jour. of Physiology*, Vol. XVIII., p. 440.

[2] Mowat: *Trans. Inst. M.E.*, Vol. XXXIX., p. 485.

ness, may have given rise to the administration of emetics. A grave danger from the use of emetics should be pointed out. There is always the chance of the patient relapsing into a comatose condition, so that insufflation-pneumonia may result, a number of cases of which have been reported.

Stimulants.

Up till quite recently, and indeed even yet in some places, it was the recognised custom in many works for the men who were engaged in work where they were exposed to gas, or in any particular work which was exceptionally dirty, to get so many glasses of whisky (four or five a day) as part of their wages, and the men would cease work every three hours or so, come up, stand in line, get their glass of whisky, and after drinking it, return to their work. The result was that many of the men were working in a semi-drunken condition. It is now known that drinkers are very susceptible to the action of poisonous gases. Happily this custom is dying out; but the habit still exists of giving a stiff dose of "neat" whisky to the patient as soon as he recovers sufficiently to swallow it. In a discussion held by the Federated Institute of Mining Engineers, one member who had been making enquiries regarding the efficacy of alcohol in gas-poisoning, mentioned a case where two men at work "sinking," were overcome by damp. One or two of their mates went to their assistance only to be overcome in their turn. A tinker who was passing pretty well saturated with drink, went down, brought up the men, and was none the worse except that he became violently sick.

But persons are affected differently by the want of oxygen. Dr Haldane, in putting persons in a closed chamber in his laboratory at Oxford, found that some were distinctly more sensitive to the want of oxygen than others. We are of opinion that alcohol in any form should never be administered in such cases, as large doses of alcohol cause marked fall of temperature. One of us having made extensive thermometric observations on "dead-drunks," invariably found subnormal temperatures. Several advocate the use of ether, but for the like reason we should not advise its employment. By far the best stimulant, perhaps, is the subcutaneous injection of *strychnine*, as it not only diminishes the loss of heat from the body, but increases the production of heat by stimulating the pulmonary and cardiac nerve-centres. At the same time hot, strong coffee not only increases the body-temperature, but has a stimulating action on the heart, and, as we have already seen, threatened syncope is one of the most pressing symptoms we have to combat.

In Germany a number of cases have been treated by *nitro-glycerine*. Hoffman[1] employed $\frac{1}{50}$th gr. as the dose in a very serious case of poison-

[1] Hoffman: "Nitro-glycerine in Illuminant and Water-gas Poisoning," *Brit. Med. Journ.*, November, 1889.

ing by illuminant-gas in a woman who was apparently dead, the respiration and pulse being scarcely perceptible. Half a minute after the injection there was a perceptible improvement of the pulse and respiration, and in half an hour she regained consciousness, and afterwards made a good recovery. Vary [1] also used ten drops of a 1 per cent. solution in a serious case of gas-poisoning, and found a remarkably rapid amelioration of the symptoms.

The *Critic and Guide* for May 1908, advised the exhibition of *peroxide of hydrogen* (given in solution by the mouth) in cases of coal-gas poisoning. Where this had been administered it had been found very efficient. The dose by mouth is about one ounce, diluted with an equal volume of water; *per rectum* about two ounces are given in full strength. It may be frequently repeated. They declare that oxygen is absorbed from the hydrogen peroxide into the blood-current.

We advise the following routine treatment:—In mild cases where the patient is conscious, very little active treatment is necessary. Hot, strong coffee may be administered, and care should be taken that the patient is kept warm and does not exert himself too soon. Next day he should get a good dose of saline purgative. In more serious cases where the patient is unconscious, oxygen is of the greatest value. The patient should be kept in the recumbent position, warmth directly applied, and vigorous friction employed. A hypodermic injection of strychnine may also be given. When possible, oxygen should be administered with a proper mask. If deeply comatose, venesection should be performed, and at the same time an intra-venous infusion of normal saline solution should be given. Intra-cellular injections may be used instead.

As regards other treatment, light, nourishing diet should be given in order to assist the organism to supply fresh corpuscles. At the same time, attention should be paid to the proper action of the kidneys and bowels, and the mixture, which is the favourite with the men when they are gassed, viz., salts and cream of tartar, may be given.

In chronic cases of CO poisoning, besides plenty of fresh air and good nourishing food, a tonic should be administered, and we have found nothing better than a combination of iron, arsenic, and strychnine.

The following is the warning notice issued by the Chief Medical Inspector of Factories in 1908 (Form 932). It must be hung in all factories and workshops where producer-gas is used for power purposes.

NOTICE.

The gas used is poisonous if breathed.

It has little or no smell.

[1] Vary: "Nitro-glycerine in CO Poisoning," *Medical Age*, Detroit, 25th February, 1891.

No man must be alone in any work likely to involve exposure to it. If in the course of his work, or for rescue purposes, he has to enter any enclosed space suspected to contain the gas, he must have a rope tied securely round his waist, held at the other end by a mate.

Ropes for this purpose are kept at . (Here is stated the place.)

Symptoms.

The first symptoms are giddiness, weakness in the legs, and palpitation of the heart.

If a man feels these, he should at once move into fresh, warm air, when he will quickly recover if slightly affected. He should avoid exposure to cold.

He should not walk home too soon after recovery; any exertion is harmful.

First Aid.

Remove the patient into fresh, warm air.

Send for the oxygen apparatus.

Send for the doctor.

Begin artificial breathing at once, if the patient is insensible, and continue it for at least half an hour, or until natural breathing returns.

Give oxygen at the same time, and continue it after natural breathing returns.

Use of Oxygen Cylinder.—Open the valve gradually by tapping the lever key (which must first be extended to its full length) with the wrist, until the oxygen flows in a gentle stream from the mouthpiece into the the patient's mouth. The lips should not be closed round the mouthpiece. The nostrils should be closed during breathing in, and opened during breathing out. If the teeth are set, close the lips and one nostril. Let the conical end of the mouthpiece slightly enter the other nostril during breathing in, and remove it for breathing out.

Artificial Breathing (Schäfer Method).

Place the patient face downwards as shewn in the diagram.

Kneel at the side of the patient and place your hands flat on the small of his back, with thumbs nearly touching, and the fingers spread out on each side of the body over the lowest ribs.

Then promote artificial breathing by leaning forward over the patient, and, without violence, produce a firm, steady, downward pressure. Next release all pressure by swinging your body backwards without lifting your hands from the patient.

Repeat this pressure and relaxation of pressure, without any marked pause between the movements, *about fifteen times a minute* until breathing is established.

CHAPTER XIV.

PATHOLOGY OF CARBON MONOXIDE POISONING.

In dealing with this part of the subject it will be necessary to consider the action of this gas upon the body, first, in acute poisoning, and, second, the action which follow upon recovery from unconsciousness after exposure to the gas.

The *post-mortem* appearances which are most likely to be found on the body after exposure to an atmosphere containing a large percentage of this gas are as follow: The skin, more or less generally, but over also the face, the front of the chest and lower front aspects of the abdomen, the insides of the thighs and insides of the forearms more particularly, exhibits a strikingly rose-red to cinnabar-red coloration; the *post-mortem* staining or hypostasis is modified in colour toward a pinkish tint, even at a time when a green colour from decomposition is developed on the right iliac region of the abdomen; and the body in some cases will, indeed, exhibit almost a life-like appearance. The same marked coloration will be observable on dissecting the body. The muscles of the chest, on being cut into and reflected for the purpose of enabling the chest cavity to be opened, show the same bright-tinted colour, and the organs of the chest and abdominal cavities, notably the lungs in the former, and the liver, intestines, and mucous membranes in the latter, the like pinkish or rose-red colour.

The blood which comes from the cut muscles, vessels, and organs, and the blood which accumulates in these body cavities after being emptied of their organs, is of a bright, cherry-red colour, is very fluid in character, and does not readily coagulate.

With regard to the facial expression of those who have died from this gas, some observers have noted a placidity of expression which they believe to indicate an absence of all struggle before death. This we have observed personally in several cases. Other observers have recorded as characteristic of this form of gaseous poisoning, the retention of the bodily heat for longer periods than usual after other forms of death, even until from ten to forty hours have elapsed. This we have not observed unless in cases in which, from the clothing of the body and its surroundings, the body-heat would in any form of death have also been retained longer than usual. We do not look upon this feature as other than incidental. The comparatively slow onset of putrefaction of

the body in this mode of death has also been remarked upon by a few observers. This, however, in our opinion, is not sufficiently marked to prove characteristic of the mode of death, but is an indication that those who have so died have either been in good health or enjoyed some measure of good health prior to exposure to the gas. At the same time, it is true that blood largely saturated with CO gas may be kept for long periods in closed glass tubes or bottles without undergoing decomposition. We have seen this in blood preserved by us for months.

The general *post-mortem* signs in this kind of death are those of asphyxia. There is general engorgement of the venous system, the chambers of the right side of the heart being engorged with blood, and the parenchymatous organs being similarly engorged. In our experience, it is rare to find the mass of blood in the heart sharing the arterial hue so characteristic of blood in the muscles and in the organs.

The question has occasionally arisen medico-legally: whether the blood of a dead body may absorb carbon monoxide gas when exposed to an atmosphere containing that gas?

The following two cases are illustrative. That recorded by Wachholz and Lemberger[1] possesses some points of interest.

The mummified body of an apparently newly-born child was found in a chimney of a dwelling-house, and it had probably been placed there by the mother after secret delivery. As infanticide was suspected, it was necessary to determine whether or not the child was alive at birth, and, if so, whether it had been placed in the chimney dead or alive. The body being in a state of mummification, no little difficulty was experienced in obtaining a solution of blood from the body, but by placing pieces of internal organs in weak solutions of potassium carbonate, a dark reddish-brown solution was got, which, on spectroscopic examination, gave the ordinary hæmoglobin spectrum, but none of CO-hæmoglobin. This result being negative, the observers conducted the following experiments. In the first set of experiments, blood taken from fresh bodies of persons who had been poisoned accidentally by CO was employed, and, also, defribrinated blood saturated with pure CO gas. Specimens of these bloods were dried in watch-glasses in a room the temperature of which was higher than usual. They were left for a time sufficiently long and in a temperature sufficiently high to facilitate decomposition. In a second set, white mice, rats, guinea-pigs, and rabbits were killed by exposure to the gas, their bodies were left to decompose and mummify. In the third set, the bodies of still-born children were placed in glass jars filled with CO gas.

In the bloods used in the first set of experiments CO could be de-

[1] *Experimentelles zur Lehre von der Kohlenoxydvergiftung. Viertelj. f. Gericht. Med.* Apr. 1902, p. 223.

tected spectroscopically, notwithstanding their exposure for two months to the ordinary atmosphere of a room. After two and half months' exposure, it was found impossible to obtain the spectrum of CO in the blood dried at the higher temperature, while after five and a half months the spectrum could be found in the blood which had been left to decompose, as well as in the samples dried at ordinary room temperatures.

In the bloods of the animals used in the second set of experiments, the presence of CO could be demonstrated spectroscopically one month afterwards.

These observers further found that in the bodies of the still-born children which had been subjected to an atmosphere of CO gas, the livid patches of cadaveric lividity in the skin changed to a red colour at the end of twenty-four' hours exposure. Half-an-hour thereafter, portions of these patches were excised, treated, and examined spectroscopically, when CO-hæmoglobin was found, but in blood taken from the hearts of these children no CO-spectrum was discovered. At the end of seven days' further exposure, however, even the heart blood yielded the spectrum of CO-hæmoglobin.

From the first set of experiments, therefore, it would seem to be established that in decomposed blood in which fresh carbon monoxide was present, the spectrum of CO-hæmoglobin may be discovered for variably long intervals of time after original exposure to this gas, notwithstanding decomposition, and, further, that in decomposed and mummified bodies of animals killed by the gas, the spectrum could be obtained after the lapse of a month. From the last set of experiments, it appears also to be established that CO gas is capable of penetrating the unbroken skin of a corpse, and of uniting with the hæmoglobin of the blood in the superficial skin vessels in cadaveric lividity. These experiments further showed, in connection with the case which prompted the investigation, that even if the observers had been successful in proving the presence of CO in the blood of the mummified child in the chimney, this would not have conclusively proved whether the child had been placed there during life or after death.

The second case was recorded by Leonpacher.[1] It also has points of medico-legal interest. At the burning of a farmhouse, the partially-burned dead body of a man fell down from an upper garret where straw was stored. The dead man had often quarrelled with his wife, and shortly before the occurrence of the fire there had been serious fighting between them, as shown by traces of blood on the floor where they had been fighting, and by the further fact that the woman had bruises on her body. The wife admitted that there had been a quarrel, and that she

[1] Leonpacher: *Munch. med. Woch.*, 1904, p. 1168.

had given her husband a blow on the head with a pot. On *post-mortem* examination of the body of the dead man there was, in addition to the lesions due to the effects of the fire, on the left side of the head between the dura mater and the skull bone, a large effusion of coagulated blood which had come from rupture or injury to the left middle meningeal artery. The brain, however, was intact. In the throat, larynx, and trachea a large amount of soot was found. The blood of the heart gave the spectrum of CO-hæmoglobin. These findings indicated that the dead man had received just before his death an injury to the skull, and while living, had been exposed to the fire in the house, had inhaled smoke as shown by the soot in his air-passages, and also CO in the smoke.

The question arose in Court, whether this man, in view of the severe injury to his head, could have been able voluntarily to climb the ladder to the garret for the purpose of setting the house on fire, or whether, when unconscious from the head injury, he had been carried up the ladder and the house set on fire by the wife to cloak her murderous attack. The possibility of occurence of the former hypothesis had to be admitted, since such supra-dural hæmorrhages sometimes proceed but slowly and, at first, cause little or no disturbance, and that it is only after an interval of some time that sufficient blood is effused to exercise such compression of the brain as to produce profound coma. During such an interval, therefore, the deceased may have climbed the ladder by his own exertions.

It will be remembered that, in the Chantrelle case, the defence was that the cause of death of the deceased wife was coal-gas poisoning from the rupture of a gas-pipe behind a window shutter. Examination of the pipe, however, indicated that it had been cut purposely to give colour to this defence, probably by the accused himself. The jury found that the deceased had died from poisoning by opium or some of its preparations.

With regard to the possibility of the blood within the superficial vessels of the skin and mucous membranes absorbing gases from the air, there can be little doubt. For example, it is well-established in cases of bodies of persons who have died from cold and exposure, and in certain cases of drowning, that the blood in the vessels of the lips and over the malar bones may become arterialised solely as the result of exposure to the air. Venous blood, when exposed to the air and especially when briskly whipped with a bundle of wires or twigs, will become rapidly arterialised by absorption of oxygen from the air.

In this connection it may be noted that Lampugnani[1] has stated that he has found that CO gas was longer associated with the blood of bodies which had been buried than with the blood of those exposed to the open air, and that in dried blood it can be detected after a longer period than

[1] Lampugnani: *Giornali di Med. Leg.*, 1899, No. 3.

in liquid blood, and much longer in the dried blood of man than in that of dogs, rabbits, and other animals.

Where it is suspected that burning has been employed to cloak the commission of a crime in some other way, the foregoing question becomes of considerable importance to the medical jurist. Such a case as this is not rare :—A man commits a murder by violence, and in order to conceal the effects of his murderous violence, sets fire to the house or building in which the victim lies. The fire may destroy some of the traces of the violence, or at least may change their character so as to shake the value of a judgment respecting their true cause. The question which the medical jurist has then to face is : Is it possible by any means to determine whether death in such a case was caused before or after exposure to the fire? Brouardel, as might be expected, has pointed out that if a person has continued to breathe even for a short time after the fire has been in progress, and in air containing carbon monoxide, that gas may be detected in the blood. This would happen, of course, whether the person had been previously rendered unconscious from violence or from any other cause. But in view of the results of the experiments already recorded, this gas found in the body may have been absorbed after death. It appears to us, therefore, that special attention ought to be given to the presence or absence of deposits of carbonaceous matter in the air-passages of such bodies, because if these are found, they are a true indication that respiration was in operation during the currency of the fire, and the conjunction of such deposits with the discovery of CO-hæmoglobin in the blood would go a long way to indicate that death was at least partly due to the effects of asphyxia by smoke and partly to CO gas therein.

Mirto has noted that where a dead body has been exposed to this gas for some time, the blood of the anterior part of the liver contained more CO gas than the blood of the posterior portion. Strassman and Schultze also drew attention to this point in 1904. They regarded the equal distribution of CO in the blood in the body as an important factor in the diagnosis of death from CO poisoning. They upheld, also, the contention of Wachholz and Lemberger that CO could be absorbed into the blood of the skin of a dead body, and that it could be detected earlier and at all times in greater amount in the superficial than in the deeper tissues. These observers were led to an investigation into this subject by the following case.[1] A woman was found in her room with one end of an indiarubber tube in her mouth and the other attached to a gas-pipe, the tap of which was open. The case had certain presumptions in favour of suicide, but on the day before the burial, the father of the deceased declared to the police that the pipe had been placed in the mouth of the

[1] Strassman and Schultz : "Untersuchungen zur Kohlenoxydvergiftung," *Berlin. klin. Woch.*, 1904, p. 1233.

dead woman in order to suggest suicide, and that he had reason to believe that, at the instigation of the husband of deceased, an operation for inducing abortion had taken place and that this had led to her death. Examination of the body, however, showed that no evidences of abortion were present, and that the cause of death was CO poisoning.

It has been stated, moreover, that this gas is to be found in normal blood, or in the blood of persons believed to be normal.

Gréhant was the first to demonstrate the presence of combustible gas in normal blood. Nicloux and St Martin demonstrated that the major portion of this gas was carbon monoxide; indeed, Nicloux[1] goes the length of saying that CO is a normal constituent of the blood of animals living in Paris.

In order to determine whether this gas was slowly absorbed from the air or was generated within the body of the animal itself, he examined the blood of a country dog, and found that the amount in it was as large as in town animals. It is well known that in populous centres large amounts of this gas are constantly being discharged into the atmosphere, yet only minute traces can be found in the blood of man or animals. Potain and Drouin[2] suggest as an explanation of this, that when CO is freely mixed with normal atmospheric air, it is slowly oxidised into CO_2.

In a later communication, Nicloux utilised the ascertained fact that after asphyxiation of an animal the quantity of CO in the blood was diminished, to aid him in proving that CO was autogenetic in the blood. He found by experiment, indeed, that dogs, which were asphyxiated to a point just short of death, only regained the amount of CO originally present in their blood in about one hour after resuscitation. These observations were corroborated in the main by Lépine and Boulud,[3] who showed further that an increased percentage of autogenetic CO exists in the blood in certain anæmic conditions, as cancerous cachexia and pernicious anæmia. They, indeed, put the query whether this gas does not exercise such a toxic effect as to be a factor in the production of the anæmia. So far as we know, no explanation or solution, except as follows, has been offered regarding the alleged presence of CO in normal blood, and in increased amounts in certain diseased states of that fluid.

Buckmaster and Gardner[4] have put these findings of Nicloux to the test of experiment. Nicloux had stated that the normal blood of dogs in Paris contained about 1·6 c.cm. of CO per litre, and that if a dog had

[1] Nicloux: *Comptes Rendus des Sciences*, Vol. 126, p. 1527; *ibid.* p. 1595.

[2] Potain and Drouin: *Ibid*, 1898, Vol. 126, p. 939.

[3] Lépine and Boulud: *Comptes Rendus de la Soc. de Biolog.*, 10th April 1905; and *ibid.* 1906, p. 302.

[4] Buckmaster and Gardner: *Brit. Med. Jour.*, 27th November, 1909.

been anæsthetised by chloroform, that the gas increased in amount to 2·5 or 2·6 c.cm. per litre. But the above-named observers, on re-investigating the question, could not discover any CO whatever in the normal blood of cats, nor in the blood of cats anæsthetised by chloroform. In the course of their experiments, however, they found that in the latter case, most of the chloroform vapour in the blood came off with the blood gases when extracted at 40°C. Noting that Nicloux's method of estimating the CO in his experiments was by passing the blood gases over iodine pentoxide at 150°C., and determining the iodine liberated in terms of CO, they repeated his experiments, with variations, and examined (1) the effect of heat on the iodine pentoxide, (2) the effect of chloroform vapour on that substance, and (3) the effect of chloroform vapour on alkalies. They then discovered that when chloroform vapour was passed over solid KHO, or was acted upon by the re-agents used by Nicloux, it was decomposed and CO was formed. Thus they concluded (1) that chloroform is not decomposed in the blood with the formation of CO, but (2) that it is decomposed into CO by the solid KHO over which the gases of the blood containing chloroform vapour were passed to rid them of CO_2.

Much has been written regarding the maximum interval of time after exposure to this gas at which CO-hæmoglobin may be found to exist in the blood of a person. This is an important matter from the point of view of litigation in compensation cases, and therefore some discussion must be made concerning it. An interesting contribution to the subject is that by Michel.[1] He mentions what has long been known, viz. :—that the combination of CO with hæmoglobin is more stable than the oxygen combination, and that the former was disestablished or disunited not only by mechanical but also by vital processes in the circulation. But the point which up till the present has not been made clear is:—Within what period of time after alleged poisoning by this gas has occurred, may the presence in the blood of CO-hæmoglobin be proved? Involved in the answer to that question is another:—How long time is required to free the blood from the gas?

Regarding the second question, it has to be pointed out that observers differ in opinion. Pouchet, for example, stated that he was able to detect the gas in the blood of surviving persons after sixty hours.[2] On the other hand, Koch[3] stated the period to be ten hours, and Hofmann,[4] two hours only.

[1] Michel: "Ueber die Dauer der Nachweisbarkeit von Kohlenoxyd im Blute und in Blutextravasaten überlebender Individuen," *Viertelj. f. Gerichtl. Med.*, 1897, p. 36.

[2] Pouchet: *Ann. d'Hyg. Pub.*, 1888, Vol. XX., p. 361.

[3] Koch: *Zur Encephalomalacie nach Kohlenoxydvergiftung*, Greifswald, 1892.

[4] Hofmann: *Lehrbuch der Gerichtl. Med.* 7 aufl., p. 706.

Other observers, however, have given different variations of time. Wesche,[1] for example, in a case in which several persons were poisoned by this gas, found in one of the victims, a woman, only a very indistinct spectrum of CO-hæmoglobin at the end of two hours after the accident, and, moreover, that the blood only responded indecisively to Hoppe-Seyler's test with sodium hydrate. This result incited Wesche to make experiments on animals regarding the duration-point. After he had convinced himself by experiments *in vitro* that the degree of combination of CO with the hæmoglobin is only a relatively weak combination, he tried to discover whether this degree of fixity of combination was the same in the living organism. Placing a rabbit in a glass chamber and allowing this gas to in-flow till the animal was in convulsions, he then took it out, placed it in fresh air for half an hour, and thereupon killed it. Examination of its blood spectroscopically and by Hoppe-Seyler's test proved negative of the presence of CO-hæmoglobin. He concluded, therefore, that this gas could not be proved to be present in blood at the end of half an hour after exposure to the fresh air. He next tried a similar experiment by exposing a second animal to the fresh air for a quarter of an hour only after removal from the gas chamber, and he met with the same negative findings. From both experiments, therefore, he arrived at the conclusion that all the CO in combination with the hæmoglobin could be dissociated and removed from the blood after fifteen minutes' exposure to fresh air.

Michel tested the accuracy of these conclusions; but he devised other means whereby the saturation of the blood of the animal by the gas was much more slowly induced, and any CO_2 gas existing in the illuminating-gas employed in the experiment was eliminated. He used cats as the animals of experiment, thinking there might, perhaps, be some difference between the action of the gas on a carnivorous animal and a herbivorous. The period of exposure of the animal to the gas in the chamber was extended to forty minutes. He was able in those instances to detect spectroscopically the presence of CO-hæmoglobin. When, however, the gas-poisoning was more rapidly induced, as in Wesche's procedure, his results corresponded with those of that observer. His general conclusion was that the longer the period of exposure to the gas, the longer was the period of time in which the CO-spectrum could be obtained, but that the length of time wherein the presence of CO in the blood may be proved, does not depend alone upon the duration of the period of exposure, but also upon individual peculiarities of animals, the influence of which cannot easily be estimated; in most cases, however, the length of time of detection always kept equal pace with the period of exposure to the gas.

[1] Wesche: "Ueber Leuchtgas-vergiftung und Kohlenoxydblut," *Viertelj. f. gericht. Med.*, 1876, Vol. XXV., p. 276.

Falk[1] propounded the view that the presence of CO could be proved after a longer period in the muscle extract of a poisoned animal than in blood itself. Michel also tested this statement, but he found that when the blood itself failed to give evidence of the presence of the gas, so did the muscle extracts.

Returning to the question of the maximum duration of time when the presence of the gas may be discovered in the blood of live animals who, after partially succumbing to the effects of CO, have been exposed thereafter to the fresh air, Michel was unable to prove the presence of the gas later than forty-one minutes after the removal of the animal to fresh air, in most instances, indeed, the period was found to vary between sixteen and forty-one minutes.

When CO as a constituent of abnormal air is inhaled, it expels the oxygen from the hæmoglobin of the corpuscles and unites with the hæmoglobin to form a more fixed combination, but the respiration thereafter of fresh air causes it to be dissociated and to be expelled from the blood, the hæmoglobin being thus enabled again to unite with the oxygen. Donders was of opinion that the process of recovery was founded merely on a simple process of dissociation. The findings of St Martin strengthened that conclusion.[2] He showed that if blood containing CO be kept standing in the open air, a small portion of the gas will disappear from it after a few hours' exposure. Jaederholm, indeed, affirms that in blood so exposed the CO entirely disappears by the end of the first week.[3]

Casper-Liman succeeded in eliminating from the blood taken from a CO-poisoned man the whole of that gas, by shaking the blood in atmospheric air for half an hour, and in finding spectroscopically the spectrum of oxy-hæmoglobin reducible by ammonium sulphide as in normal blood.[4]

Dresser[5] showed, as the result of experiments, how far in an atmosphere containing CO, the blood of an individual exposed thereto exhibited decrease of oxy-hæmoglobin and increase of CO-hæmoglobin, and also how rapidly recovery of the blood succeeded exposure to fresh air. In a rabbit so exposed, and immediately after the onset of convulsions from the gas, the blood on examination showed 50·1 per cent. only of oxy-hæmoglobin after fifteen minutes' exposure to fresh air, that after twenty minutes'

[1] Falk : "Zur Casuistik der Kohlenoxyd-vergiftung." *Viertelj. f. gericht. Med.*, iii., Bd. II., 1891, p. 263.

[2] St Martin : *Comptes rendus de l'Acad. des Sc.*, Vol. CXII., p. 1232.

[3] Jaederholm : "Die Gerichtlich-medicinsche Diagnostik der Kohlendunstvergiftung." *Norsk. Med. Arked.*, Nos, 11 and 12, 1874.

[4] Casper-Liman. *Lehrbuch der Gerichtl. Med.*, 1876, p. 570.

[5] Dresser : "Zur Toxicologie des Kohlenoxyds," *Arch. f. exper. Path. u. Pharm.*, Bd. XXIX., 1891, p. 119.

exposure the blood showed 73·63 per cent. of oxy-hæmoglobin, and after little more than two hours the blood of the animal was restored to the normal. When a third blood-test was made two hours and fifty minutes after the first, the oxy-hæmoglobin proved to amount to 91·5 per cent.

By experiment, therefore, as well as in actual experience, it is well-established that not even in the most severe poisoning by this gas does the blood become entirely bereft of oxygen, that even when the period of poisoning was protracted and the degree of saturation high, there still remained about 20 per cent. of the blood as oxy-hæmoglobin. There is never, therefore, complete saturation of the hæmoglobin with CO gas. After colliery explosions, the degree of saturation of the blood with this gas has been found in certain bodies to be as high as 80 per cent.

What has been found experimentally, therefore, in animals, may be taken within limits as similar in human beings, although it has to be borne in mind that the pulmonary gaseous interchange in small animals is much more rapid than in man, from which would likely follow the possibility of a longer period, even after exposure to fresh air, within which the presence of CO in the blood could be detected. Besides, other factors operating in different cases cannot be left out of the calculation, such as the percentage of CO present in the air of exposure, and the differing susceptibilities of animals in different classes and of animals, including man, in the same class. In addition to these may be placed the personal equation of the observer and the sensitiveness of the methods of examination.

It is only such conditions as the foregoing which will account for the divergent results given by observers as to the times at which the gas could be discoverable and has been discovered in the bodies of those exposed to its influence. In one case, for example, it is recorded that, although the victim died at the end of five and a quarter hours, no CO could be found by any method of testing used. In another, recorded by Casper-Liman,[1] in which three persons, one found unconscious, two of them recovering, and the third dying on the following day, the blood of the dead man showed normal conditions, nor could the gas be found in his blood during his life after nine hours. Chlumsky[2] has reported the poisoning of a mother and two children after exposure to this gas. One of the children, aged nine months, died at the end of fifteen hours. Spectroscopic examination of the blood of this child failed to reveal the presence of CO, but on examination of the blood of the other child a positive result was obtained. In a case recorded by Michel,[3] which

[1] Liman: *Loc. cit.*, p. 581.

[2] Chlumsky: "Tod im Kohlenoxyd und tod durch Kohlenoxyd," *Viertelj. f. gericht. Med.*, 1893, Bd. V., p. 321.

[3] Michel: *Op. cit.*, p. 45.

occurred in Vienna, a man, aged 44 years, with his wife and a daughter of ten years of age, were poisoned by the gas. The wife only was resuscitated, although four hours later she died. No CO was found in the blood of the wife, but it was found in the blood of the man and the daughter. In this woman's case, it appears as if the respiration of fresh air for four hours had been sufficient to dissociate the CO from the blood. Posselt[1] publishes a case in which a woman died at the end of seven days after exposure to CO gas. Two days after the accident, the presence of the gas in the blood was proved spectroscopically, and the blood responded also to the Hoppe-Seyler and Kunyiosi Katayama chemical tests. Especially instructive, also, is a case recorded in the Reports of the Vienna Forensic Institute. A stoker, 45 years of age, was found unconscious on 7th February at mid-day. At first he was thought to be "dead drunk," therefore he got no active help until the following day. As by that time he did not seem to be improving, he was taken to hospital, but he died on 9th February at 4 p.m. Spectroscopic examination of the blood made forty hours after finding the man, did not reveal the presence of CO gas.

Such examples as the foregoing will, perhaps, suffice to show the variability respecting this limit of time within which detection may be made of the gas.

Summing now the available evidence respecting this time of duration of CO in the blood after exposure thereto and exposure thereafter to fresh air, the burden of proof goes to indicate that the time is limited to three or four hours at the most, when the patient survives longer than that period of time.

But with regard to the presence of CO in extravasations of blood in persons who have been exposed to the gas, it seems clear that this gas may be detected for a much longer period in such extravasions than in circulating blood. Michel experimented respecting this point on animals, and he found that in extravasated blood the spectrum of CO-hæmoglobin might be found till the fifth day after the exposure of the person to the gas, and long after the gas had disappeared from the blood in circulation. That it disappears in time from extravasated blood goes without saying, but the rate of disappearance would seem in some measure to depend upon the rate of absorption of the extravasated blood. This corroborated the previous findings of Szigeti,[2] who was not able to discover CO in the blood of a man who survived $5\frac{1}{4}$ hours, but was able to demonstrate its presence in the blood of extravasations in his body.

One of the interesting questions at present in issue is: What is the

[1] Posselt: "Ein Fall von Kohlendunstvergiftung," *Wien. klin. Woch.*, 1893, Nos. 21 and 22, p. 377; p. 399.

[2] Szigeti: *Viertelj. f. gericht. Med.*, 1893, p. 64.

rôle played by carbon monoxide in producing death in acute cases? On this subject there are two schools of opinion. The chief exponent of the one is Haldane, who holds that death is produced by anoxæmia, while it is affirmed by the exponents of the view of the other school that death is not so much the result of diminished supply of oxygen as the result of a specific action of carbon-monoxide upon the nervous system.

Haldane's views are that all the symptoms of carbon monoxide poisoning, primary and secondary, are referable simply to want of oxygen, and he supports these views by citing the fact that if animals are placed in oxygen under a pressure of from one to two atmospheres, and carbon monoxide gas driven in on top of the oxygen, these animals continue to live notwithstanding the fact that their blood and tissues are saturated with the latter gas. But it has to be pointed out that such animals receive from the high-pressure oxygen enough of that gas in simple solution in the arterial blood to supply the tissues, in spite of the presence of the carbon monoxide. It is true to affirm that the proximate cause of death by carbon monoxide gas is asphyxia, just as it would be if death resulted from the respiration of oxygen-less air, or air laden with carbon dioxide gas. Linossier,[1] as Mott has quoted, showed that when animals are placed in an atmosphere of carbon monoxide, they died more rapidly and did not exhibit the same symptoms as when simple asphyxia was induced by placing them in an atmosphere of physiologically inert nitrogen; and that snails, whose blood does not contain hæmoglobin, when placed in an atmosphere of hydrogen or nitrogen containing the same proportion of oxygen as in a mixture of carbon monoxide and oxygen, died quicker in the latter than in the former. It has also been pointed out by Lamic[2] that frogs are capable of living for three and a half hours in nitrogen, but only for one and a half hours in carbon monoxide.

Some observers declare that carbon monoxide exercises a specific action upon the central nervous system through the circulating blood which carries it thither. In support of that belief, the lesions of the central nervous system consequent upon inhalation of the gas followed by death are demonstrable without doubt. Further attention, however, will be given to this aspect on a later page. In the meantime, the opponents of the anoxæmia theory of Haldane declare that if this theory explained all the condition, the exhibition of oxygen gas should, in case of survivors to exposure to carbon monoxide, prove more frequently successful in restoration than is realised in practice. It cannot be accepted that carbon monoxide acts on the blood exactly as does nitrogen or hydrogen or any inert gas. Of course, it may be explained

[1] Linossier: *Lyon Méd.*, 1889, No. 28.
[2] Lamic: *Thèse de Bordeaux*, 1891.

that owing to the action of the gas the brain, heart, and lungs are sufficiently crippled by the want of oxygen as not to be able to make use of oxygen when artificially exhibited.

It appears to us that a more reasonable explanation of the action of the gas upon the bodily organism is, that negatively the organism suffers from the relative anoxæmia which is consequent upon the excess presence of CO in the air, but it also suffers positively from the action of this gas upon the blood, and through the blood upon the central nervous system.

When the time of exposure to carbon monoxide is not longer than is required to produce unconsciousness in those exposed, and thus where resuscitation is possible either by the removal of the patient to the fresh air or by the aid of oxygen, or by both agencies combined, death may not be avoided because of the direct injury of the gas in the blood upon the central nervous system, whereas if the state of the patient were the sole result of anoxæmia, the continuation of the fresh air and oxygen treatment ought to suffice to bring about recovery.

Geppert,[1] as the result of experimental research, came to the conclusion that the action of CO upon the organism was not that of lessened oxygen-supply to the tissues owing to the combination of carbon monoxide with the hæmoglobin, but a specific action on the nervous system, since the type of respiration which follows the exhibition of CO gas differs materially from that observed in oxygen-starvation due to slow asphyxiation.

Claude Bernard was the first observer who found that in CO poisoning the oxygen was expelled from the blood by replacement with the former gas. This discovery led to the theory that CO acts by producing asphyxiation, but did not raise the question whether or not it produced any action specific to itself. But it was known that in CO poisoning there is no dyspnœa as there is in ordinary anoxæmic asphyxiation, since in the latter the organism seeks by increased and forced respiration to obtain more oxygen by increased pulmonary action. At first, this was looked upon merely as evidence that as CO poisoning is slower in its operations, the replacement of the oxygen in the corpuscles being a more gradual process, the demand for more oxygen was hence less urgent.

Geppert, by experimental research on animals, sought a solution of the question: How does respiration proceed in slow displacement of oxygen by CO gas in the air respired? First of all he measured the breathing of an animal in the usual way. Tracheotomy was then performed, and the tracheal cannula connected by tubes with valves to allow of separation of the inspired air from the expired air. In connexion with one of the valves a gas-meter was placed, by which could be re-

[1] Geppert: *Deutsche Med. Woch.*, 1892, 12th May, p. 418.

corded and noted the quantities of air respired per minute. Slow poisoning by CO was then produced by allowing the animal to breathe air from which the oxygen was gradually diminished in amount. Such an experiment, however, does not seem to get nearer a solution of the above problem, because whether the inspired air contains less than the normal amount of oxygen or contains CO in place of the diminished oxygen, the issue is the same, viz. :—tissue-respiration is in each case rendered more difficult, and in both cases the oxygen-content of the blood diminishes, and with it, the amount of oxygen available for the tissues.

It remained, then, to examine whether, when the tissues were supplied in the same insufficient manner with oxygen, the breathing would be affected in the same way. To achieve this, it was essential that respiration and the consumption of oxygen should be determined simultaneously. That was done in the following manner, viz :—A tracheal cannula having been placed in position and connected by two tubes to a glass vessel of 10-15 litres capacity, valves for inspired and for expired air were adjusted to these tubes. The expired air was made to pass through bottles to absorb the carbon dioxide, and an estimate was made of the amount of oxygen consumed. It is obvious that the poorer in oxygen the inspired air is, the relatively richer in nitrogen will the air become. As the result of this experiment, it was found that the respiration changed in character very distinctly so soon as the decrease of oxygen in the inspired air amounted to between seven and eight per cent. When the oxygen amount was lessened to as much as three to four times less than the normal, the respirations at once became increased in number and deeper in character.

The experiment further showed that the consumption of oxygen by the animal fell progressively, but that the rate of breathing increased sooner than did the decrease of oxygen consumption. When, however, an animal was poisoned with air containing 9·5 per cent. of CO, these changes proceeded so slowly that the individual stages of the change could easily be followed, and it was found that they proceeded much more quietly than in slow asphyxiation produced by the respiration of oxygen-impoverished air. In this case, it will be noted that after a certain time in the currency of the experiment, the oxygen consumption also fell, and, indeed, became as low as in asphyxia, but the breathing was different. The animal here breathed almost in a normal manner, sometimes a little stronger, sometimes a little weaker.

Occasionally a different result was observed, viz., the number of respirations were much increased, but each respiration was very much shallower than normal, contrary to what is found in slow asphyxiation by oxygen-impoverished air. It would appear, then, in CO poisoning as if the respiratory centre did not respond to the want of oxygen by

increase of respiration, and it is a legitimate inference from this that CO has some particular specific action on the nerve centres and nerve tissues.

It is now necessary to consider what changes, if any, are found on the bodies of those who have died from the effects of exposure to an atmosphere containing CO gas, particularly with reference to the central nervous system.

From the symptoms which are observable in cases after recovery from the acute effects of the poisonous gas, lesions of the brain and spinal cord might be predicated, but as these symptoms have already been exhaustively treated it is not necessary to recapitulate.

In a paper by Hedren[1] there is the following description of the lesions in the central nervous system of a case which came under his observation :—(*a*) numerous small hæmorrhages in the pia mater of the brain and spinal cord ; (*b*) small bloodless patches throughout the entire spinal cord, in some of which actual softening had occurred ; (*c*) thrombi in small blood-vessels of the optic thalamus ; (*d*) fatty degeneration of the endothelium of the small blood-vessels of the central nervous system ; (*e*) chromolytic and atrophic changes in the large motor cells of the spinal cord ; (*f*) partial thickening of the pia mater of the brain ; and (*g*) degenerative changes throughout the nervous system generally. Von Sölder[2] narrates the facts of a case in which, four weeks after poisoning by this gas, the patient developed progressive dementia, paralysis and atrophy of the muscles of both legs, with disappearance of the patellar reflexes and decrease of electrical excitability of the muscles. On *post-mortem* examination, serious degenerative neuritis of part of the peripheral nerves, hyaline degeneration of the vessels to the muscles, degeneration of the muscular fibres, and of the anterior cornual cells in the cervical and dorsal and, to a lesser degree also, in the sacral portions of the spinal cord, were found.

Runeberg[3] reports three cases of poisoning by the gas, two of which proved fatal. In one of these there were found extensive areas of softening in the lenticular nucleus on both sides of the brain, and in the other there were also similar areas in the brain and spinal cord. This observer attaches importance to these lesions, in relation to consequent serious mental disturbance and nervous disorders sequent to the toxic effects of this gas. He believes that the existence of such lesions indicates clearly that the toxic influence cannot be explained solely or altogether by mere displacement of oxygen from the blood. Knecht[4]

[1] Hedren : *Nordiskt. Med. Ark.*, 1912, No. 20, p. 3.

[2] Von Sölder : *Jahrb. f. Psychiatrie*, 1902, Bd., XXII., p. 287.

[3] Runeberg : *Finske Läkersallsk. handl.* XLIV., 1902, p. 495 ; *Centralb. f. innere, Med.*, Aug. 1903, p. 791.

[4] Knecht : *Deutsche Med. Woch.*, 1904, No. 34, p. 1242.

emphasises the fact that this gas exercises its poisonous effects on the brain, spinal cord, and peripheral nerves. In his first case, in which there was hemiplegia with motor aphasia, a diseased area was found in the left central area of the brain, but whether this was of a hæmorrhagic or encephalo-malacitic nature was not clear, because this patient had contracted syphilis several years before. With regard to the brain area in question, it was difficult to say how far it extended into the internal capsule. In his second case, the conditions were more complicated. There was an area of disease in the left cerebral hemisphere, which caused on the fifth day, a passing aphasia and also hemiparesis of the entire right side of the body, with vasomotory and sensory disturbances, these last betraying themselves by the characteristic disturbance in the stereognostic sense in the fingers. As the hemiplegia disappeared very quickly, except in a very small part, it was justifiable to conclude that only a small area of the brain had been originally affected.

In an old woman, who had been unconscious for four days after exposure to the gas, who after regaining consciousness was found to be imbecile, and who died at the end of twenty-three days, Cramer[1] found in the brain cortex degeneration of the fibres which was extensively distributed, and in addition vacuole formations in the ganglia cells, hyaline degenerative changes in the vessels of the basal ganglia of the pons Varolii and of the medulla oblongata, as well as diffuse proliferation of the glia. In the case reported from the Vienna Forensic Institute, *post-mortem* examination revealed symmetrical softening of the lenticular nuclei.

Rokitansky[2] has reported the case of a young woman who died nine days after being poisoned by the gas, never having regained consciousness in the interval. *Post-mortem* examination of her body revealed double broncho-pneumonia, enteritis with some peritoneal hæmorrhages, and thrombosis of the veins of the left lower limb; hyperæmia and œdema of the brain; softening of the grey matter of the spinal cord, punctiform hæmorrhages being distributed throughout its area and especially in the neighbourhood of the anterior cornua; and reddish-grey softening in the cervical and in parts of the dorsal portions of the cord. The investing membrane or sheath of the sciatic nerve and its branches were coloured red owing to engorgement of the vessels.

Two of the most important recent contributions on this subject are those which have been made by Schaeffer and Mott, and to these we shall devote some attention.

Schaeffer[3] is of opinion that more minute histological examination of

[1] Cramer: *Centralb. f. Allgemeine Path.*, July, 1891, p. 545.
[2] Rokitansky: *Wien. Med. Presse*, No. 52, 1889.
[3] Schaeffer: *Wien. klin. therap. Woch.*, 1903, p. 1227.

brain tissues in such cases will show the presence of hæmorrhages and areas of softening in the cortex to be much more common than has hitherto been supposed, and that especially where death has occurred shortly after exposure to the gas, such histological examination is likely to throw valuable light on the diagnosis. He gives a full account of two cases in which he made such an examination of the nervous system. The first case was that of a woman, 60 years of age, who died after being comatose for forty hours after exposure to the gas, in whose body he found hyperæmia and œdema of the brain and its membranes and symmetrical areas of softening of the size of a bean in the lenticular nuclei. There was a moderate amount of arterio-sclerosis present. The second case was that of a man, 43 years of age, who died after being comatose for eighty hours. In the brain were found extensive softening and hæmorrhagic infiltration of the central ganglia, a hæmorrhage into the left ventricle, and numerous isolated and confluent hæmorrhages, about one centimetre in size, in the frontal and occipital lobes. There were, also, marked hæmorrhages into the membranes of the spinal cord, and multiple hæmorrhages into the tissues of the lungs, liver, stomach, intestines, and kidneys. In both of these cases, CO was demonstrated in the blood.

On more minute examination of preparations from both the areas of softening and parts of the brain in which there had not been hæmorrhages, he found that the blood vessels were dilated with blood cells and blood clot, and that in preparations from the brain of the second case there was marked dilatation of the finest blood vessels of the cortex, which looked as if the dilatation had been produced by artificial injection. There was, in addition, dilatation of the perivascular lymph channels and bleeding by diapedesis. He notes that of twenty-five cases previously recorded in which areas of brain softening were found, the lenticular nucleus was regarded as the part most frequently affected, but he is of opinion that other parts are equally affected although they have not been observed. He does not think it proved that the internal nuclei have a special predilection for CO poisoning, even when the peculiar anatomical arrangements of this part, which favour the onset of stasis and capillary thrombi, are taken into consideration, nor that it is merely accidental that in two or three cases subjected by him to minute histological examination, areas of softening in the cortex have been revealed.

After treatment with Marchi's osmium staining method, he found in sections of the brain outside the areas of softening in the first case, and in the whole of the peripheral and central nervous system of the second case, a general blackening of the nerve fibres in addition to an increase in size and a varicose or rosary-like condition of these fibres, thus indicating generally the presence of necrosis of nerve tissue. This osmium reaction

was found also in sections from the vagus nerve. This reaction indicates that the nerve marrow had undergone fatty change, since normal nerve tissue when treated with Müller's fluid and the osmium stain does not blacken. Seeing, therefore, that the above-mentioned changes are found in the nerve tissue dissociated from hæmorrhagic extravasations, it would appear as if these changes were brought about directly by the action of the gas on that tissue, and as if they were of a necrotic character with formation of fatty substances. In more advanced stages the nerve fibres seem to decay, and a small round-cell formation to take their place, this being probably aided by the dilated state of the vessels by thrombi, displacement of vessels, and hyperlymphosis. This destruction of the fibre can sometimes, indeed, be demonstrated in sections from the extra-spinal roots of nerves.

Schaeffer believes that when death occurs soon after CO poisoning, and where spectroscopic examination of the blood fails to reveal CO-haemoglobin, the histological examination of the central nervous system will probably contribute much toward a correct diagnosis. He has arrived at the view that the numerous hæmorrhages found in the brain and nerve tissue can be accounted for by the supposition that the action of this gas is somewhat analogous to that in ferment intoxication, and that owing to such a toxic ferment the chemical composition of the blood undergoes changes, one of which is to produce coagulation of the blood in the vessels into homogeneous masses, ferment thrombi having the same characteristics. This theory has been upheld by Kionka,[1] but has not as yet secured general adherence.

The other view, more generally held, of the intimate cause of these multiple hæmorrhages, is fatty degeneration and arterio-sclerosis of the vessels due to the action of the gas. Reference has been made (p. 97) to a case which has come under our observation, in which most marked arterio-sclerosis followed exposure to this gas.

The view that CO may directly and primarily injure the nerve elements in the central nervous system is maintained also by many observers, among others Kobert, Geppert, and Runeberg, although they do not adduce histological proof of their contention.

Mott, in a valuable contribution to the subject, entitled "Carbon Monoxide and Nickel Carbonyl Poisoning,"[2] has exhaustively examined the intimate pathological appearances found in the central nervous system of persons who have died from the effects of exposure to that gas and to the fumes of nickel carbonyl, and has correlated the lesions found with the symptoms exhibited during life in cases which had a fatal termination. His personal observations of the effects of CO gas on human

[1] Kionka : *Real. Encyl. der Gesammte Heilk.*, 1897, XII.
[2] Mott : *Arch. of Neurology*, Vol. III., 1907, p. 247, *et seq.*

beings consisted in the clinical observation during life, and the *post-mortem* examination of the body of a woman who committed suicide by illuminating-gas, and thereby caused the deaths of two of her children who occupied the same room with her, and in the pathological examination of the brains of two men who succumbed after exposure to the gaseous fumes from nickel carbonyl.

The facts of the case relative to the death of this woman and her two children are briefly these :—She was admitted into Charing Cross Hospital, London, under the care of Dr Mott, on 12th January, 1906. She had come to London the day previously with her chidren and had taken a room at a hotel. She retired to her room with the children about 11.30 p.m., and next morning, although called more than once, no response was given. The door of the room was then forced open, when it was found to be quite full of illuminating-gas, all the openings being stopped up, the burner of the gas-bracket taken off, and the gas full on. On the bed were found the two children, who were dead, and the woman herself in an unconscious condition. On admission to the hospital she was comatose, and was breathing stertorously. There was a reddish-pink colour about the face and lips. The pupils were contracted almost to pin-point size, but there was a sluggish reaction to light, and the corneal reflexes were present. Her temperature was 99·4°F., the respirations numbered 18, and the pulse 116, which was regular and of good volume. Her stomach was first lavigated with a solution of potassium permanganate, and an injection was administered hypodermically, on the possibility that she might have taken opium. Oxygen was freely administered, and as the respiration ceased occasionally, artificial respiration was then resorted to. Her arms and legs became quite rigid, the knee-jerks were diminished, but the plantar extensor reflex was present. Examination of the blood from a finger-prick was made, and the blood when diluted with water gave a pinkish colour, and on spectroscopic examination yielded the spectrum of CO with an additional band of reduced hæmoglobin. She died at 2.30 a.m. on 16th January, the interval of time between the inhalation of the gas and her death amounting exactly to four days, during the whole of which time she remained unconscious.

The *post-mortem* examination of the body showed that it was that of a well-nourished woman. Slight cadaveric lividity was present in the dependent parts, and the skin generally had the appearance of old wax. The right pleura was obliterated by old adhesions. The pericardium contained about half an ounce of blood-stained serum. The large bronchi were injected and contained a little blood-stained mucoid secretion. The left lung showed marked hypostatic congestion at the base, the right lung a patch of early pneumonia, about the size of an orange, at the posterior aspect of the lower portion of the upper lobe, and

the adjacent upper portion of the lower lobe showed consolidation due to early pneumonia. The heart-muscle was flabby, the muscle of both ventricles showing extensive fatty degeneration. The rest of the thoracic parts were healthy.

The substance of the liver was markedly fatty. The cortex of both kidneys was much injected, and their substance flabby and cloudy. The rest of the abdominal organs were healthy.

Portions of various internal bodily organs, as the liver, heart, and kidneys, were placed in Marchi's fluid, and, after hardening, sections were made and examined with the following results :—The liver showed fatty degeneration, every cell containing black-stained particles; the heart-muscle exhibited very marked fatty degeneration, every fibre indicating degenerative change in the early stages; and the kidneys showed similar changes, although not so marked as in the liver.

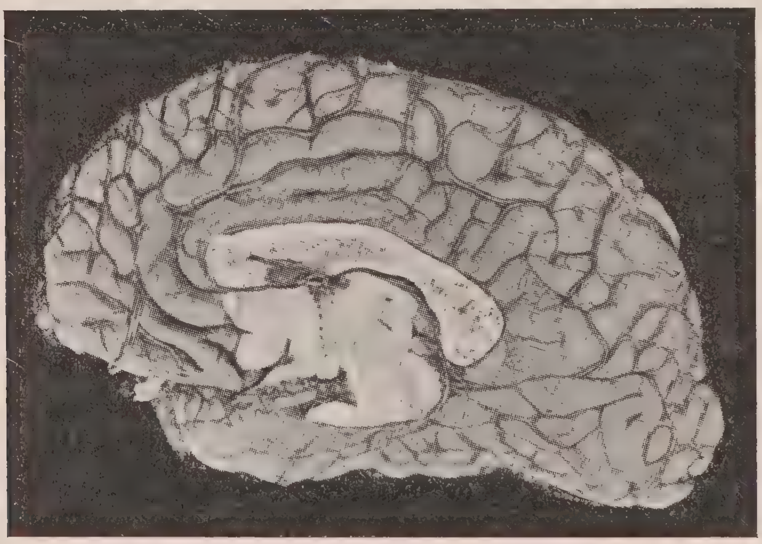
The following were the appearances found on examination of the brain :—The pia arachnoid was not thickened. There were various congestive patches about the external and mesial surfaces of the hemispheres, and all the indications of sub-pial hæmorrhages. On slicing the pons in its lower part congested vessels were seen, which suggested thrombosis or minute hæmorrhages. There were no hæmorrhages visible to the naked eye in the medulla. The right hemisphere on being dissected showed throughout the white matter, especially in the internal and external capsules and the corpus callosum, aggregations of hæmorrhagic points of varying size, which suggested either thrombosis of the perforating arteries or hæmorrhages into their sheaths, or probably both. Throughout the white matter of the centrum ovale were punctiform hæmorrhages, which became especially distinct after hardening the left hemisphere in 5 per cent. formalin and then slicing it in vertical sections.

The brain substance was then examined, with the following results :—The vessels of the cortex were found dilated, but it was chiefly in the sub-cortical white matter that the greatest amount of congestion and hæmorrhage and rupture of small vessels were found. The rupture sometimes was limited to the perivascular sheath, and sometimes into the substance of the brain itself. Many of the small vessels gave indications of glia cell proliferation and leucocytic infiltration of their sheaths. The endothelial cells of the capillaries appeared swollen, and as if division of their nuclei was taking place. Stained by Marchi's fluid, these showed degenerative changes in the walls, indicated by black staining. The most marked vascular degeneration and hæmorrhage were found in the white matter of the centrum ovale, and especially in the white matter of the occipital lobe. The vessels of the medulla oblongata were intensely congested, and in the sections stained with polychrome and eosin the red corpuscles were found to take the blue stain instead of the red. There were very

few hæmorrhages, but those that were observed were into the sheath of the vessels and not into the substance of the brain. The ganglion cells showed little alteration in form, but there was a general diffuseness of

From case of Coal-Gas Poisoning.

Fig. IV.—Showing hæmorrhages in the white matter of the brain, and especially in the corpus callosum (Mott).



From case of Coal-Gas Poisoning.

Fig. V.—A section of the brain showing punctiform hæmorrhages in the corpus callosum (Mott).

staining which may have been the result, however, of the high fever exhibited by the patient during life.

We may contrast and compare with the foregoing, those found on examination of the brains of two workmen who had succumbed to the

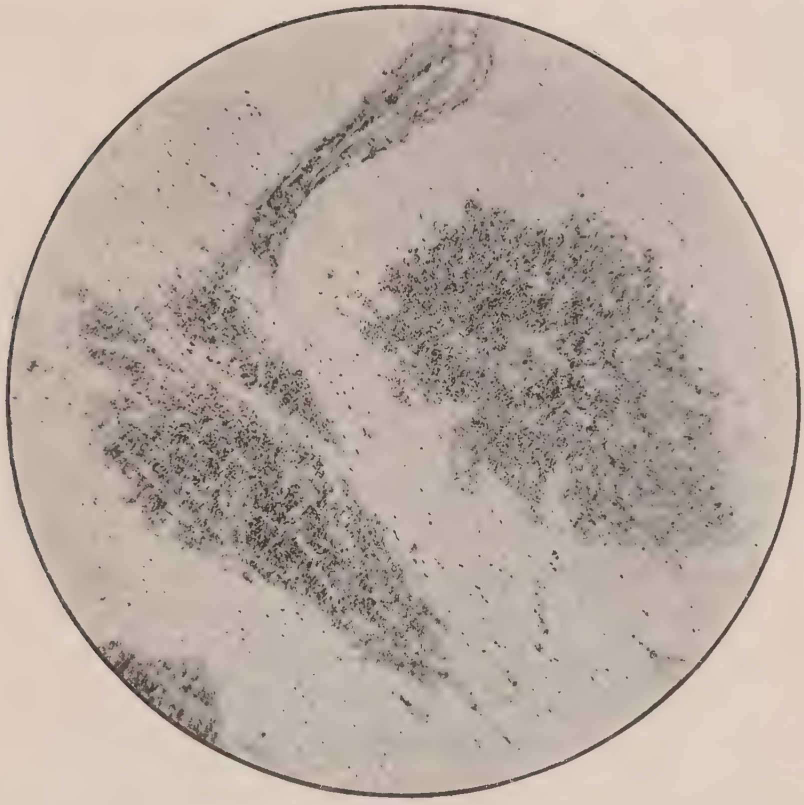


Fig. VI.—Section from brain showing rupture from small blood-vessel with hæmorrhagic extravasation into the tissues. 90 diameters. (Mott).

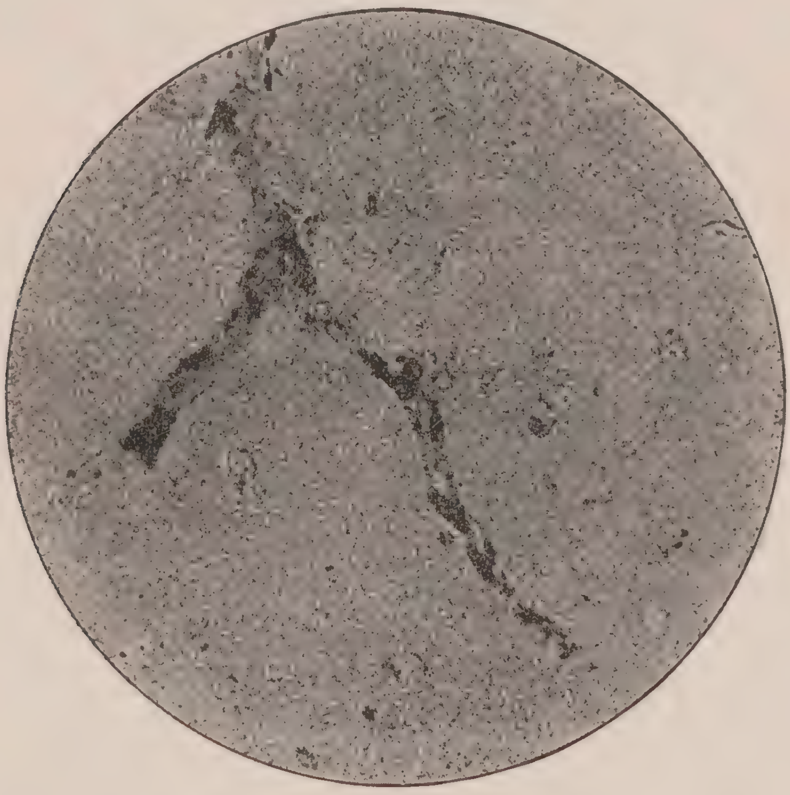


Fig. VII.—Photomicrograph of brain tissue showing fatty degeneration of the walls of small blood-vessels. The black staining by the Marchi method indicates the amount of change. ×100. (Mott).

effects of the fumes from nickel carbonyl in the Nickel Carbonyl Works, Clydach :—

1. This brain, cut in slices, had been forwarded to Mott in a jar containing spirit, and having been so hardened, the methods of examination available were limited to the Nissl staining process to show changes of structure. Portions of different parts of both hemispheres, including the motor area, the internal capsule, centrum ovale, basal ganglia, with portions of the pons and the medulla, sectioned at several levels, were taken for examination. Ten portions of brain in all were subjected to examination. Generally speaking, the blood-vessels throughout the hemispheres were engorged with blood, and the brain substance beset with capillary hæmorrhages, varying in size from the point to the head of a pin. These were most marked in the sub-cortical white matter. No hæmorrhages into the pons and medulla were discernible to the naked eye.

After embedding in paraffin and staining by the Nissl method or a modification thereof, the sections made from the ten portions of brain, amounting in number to several hundreds, were examined microscopically, and from their examination the following facts were ascertained :—(*a*) the cells of all the specimens showed some chromolytic changes, less marked, however, in the cortex than in the pons and medulla ; (*b*) the large Betz cells of the cortex were nearly normal in appearance ; (*c*) hardly a healthy cell was found in the following nuclei, viz. : the vagus, nucleus teres, spinal accessory lying in the floor of the fourth ventricle ; and the nucleus ambiguus also showed marked changes, indicating early organic change in the form of coagulation necrosis.

As the autopsy in this case was not made until forty-eight hours after death, Mott was unable with certainty to say how far these changes might not in part or wholly have been due to *post-mortem* change. The state of the small vessels and their contained blood suggested the probability, however, that the poison had affected especially the nuclei above-named. The neighbourhood of these vessels showed endothelial proliferation, and contained relatively an enormous number of leucocytes, of which the polymorpho-nuclear were by far the most abundant. The vessels in the medulla showed only broken-down corpuscles. There was general venous engorgement, but no hæmorrhages into the substance of the pons or medulla, although occasional hæmorrhagic extravasations into the perivascular sheaths were found. The hæmorrhages into the brain were more numerous than Mott had ever previously seen. They seemed to be due to rupture of the small capillary vessel walls, and were apparently mostly of recent origin. In these extravasations the corpuscles appeared to be morphologically perfect, or had formed into a red amorphous mass, while occasionally the blood was found to be

covered with very minute brownish, orange-coloured crystalline bodies, many of them tetrahedral in shape. From this examination, Mott concluded that there had probably been an acute fatty change of the capillary vessel walls produced by the poison. Owing to the method of hardening in this case, the osmic reaction could not be obtained.

Chemical analysis of about 400 grammes weight of this brain substance gave a negative result as to the presence of nickel.

2. The history of this case was briefly as follows :—This man was first seen on 20th May. He took to bed on the night of the 21st, but his consciousness was not affected until the night of the 24th, when he showed slight rambling, his temperature being then 103.8°F. On the 26th he was semi-unconscious, but was easily roused, responded to

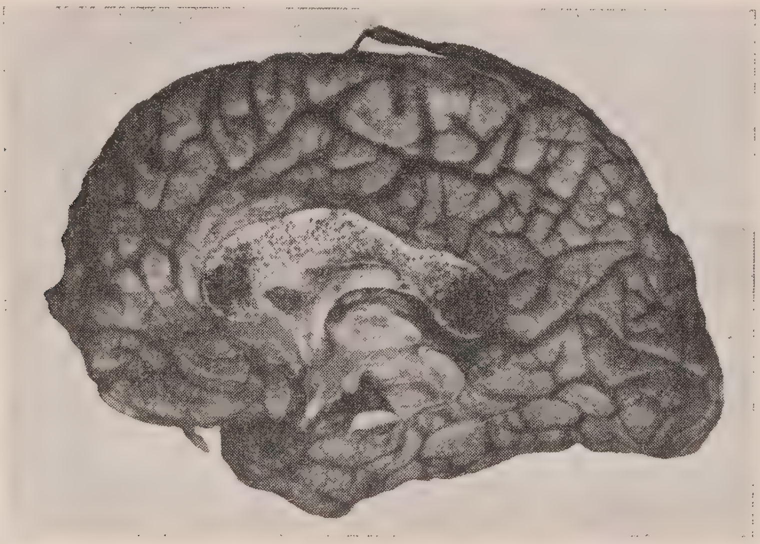


Fig. VIII.—Photograph of the mesial surface of the right hemisphere of the brain of case 2 from Nickel Carbonyl Works. Showing the hæmorrhages in the cut surface of the divided corpus callosum. (Mott).

questions, and recognised those around him fairly quickly. He remained in this state till within twelve hours of his death, when the coma became more profound, death occurring at 10.15 on the 18th. Throughout the illness there had not been any orthopnœa, but the respirations numbered sixty per minute and the type of respiration was diaphragmatic. There was no paralysis, the deep reflexes being present up till the end. Ankle clonus was got on the last two days. Plantar reflexes were greatly exaggerated, and were present until eight hours of the death.

The interval which elapsed between the time of death and the placing of the brain in formalin solution was eight hours. The organ was received by Mott on 29th May.

Examination gave the following results :—*General Appearances.*—The pia-arachnoid of the spinal cord, and base of the brain especially, had a

rusty appearance. The hemispheres were soft, but the cortical substance seemed to be firm. The hemispheres were divided in the middle line. The whole of the cut surface of the corpus callosum showed punctiform hæmorhages, its two extremities exhibiting very little white matter by reason of the number of these hæmorrhages. With a hand-lens it was observed that the blood was bright-red in colour, and had apparently escaped into the perivascular sheaths and the surrounding tissues. To the naked eye the grey matter showed little change, but with the lens hæmorrhages became visible. The peduncles of the cerebellum and the white matter of the lateral lobes in less degree gave evidence of congestion of vessels with hæmorrhages.

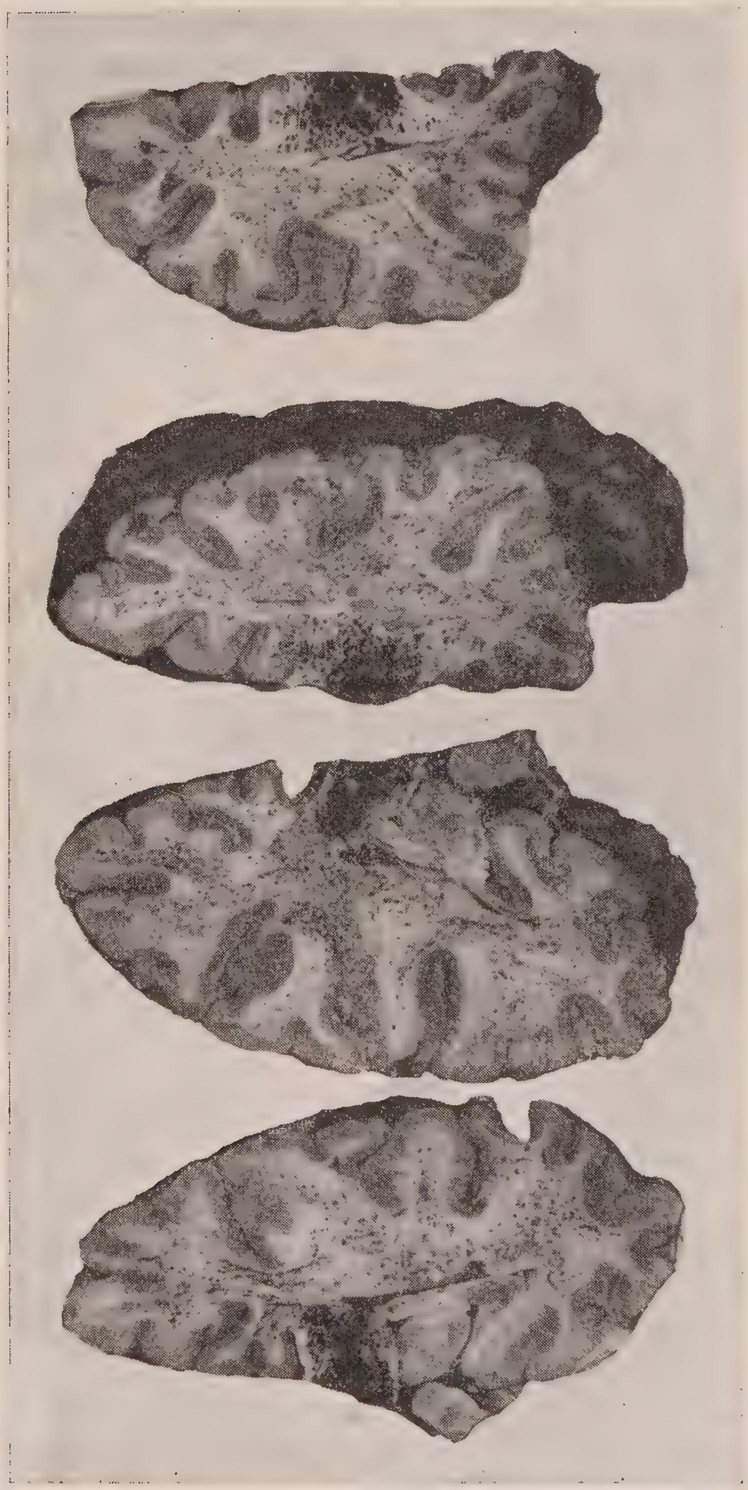
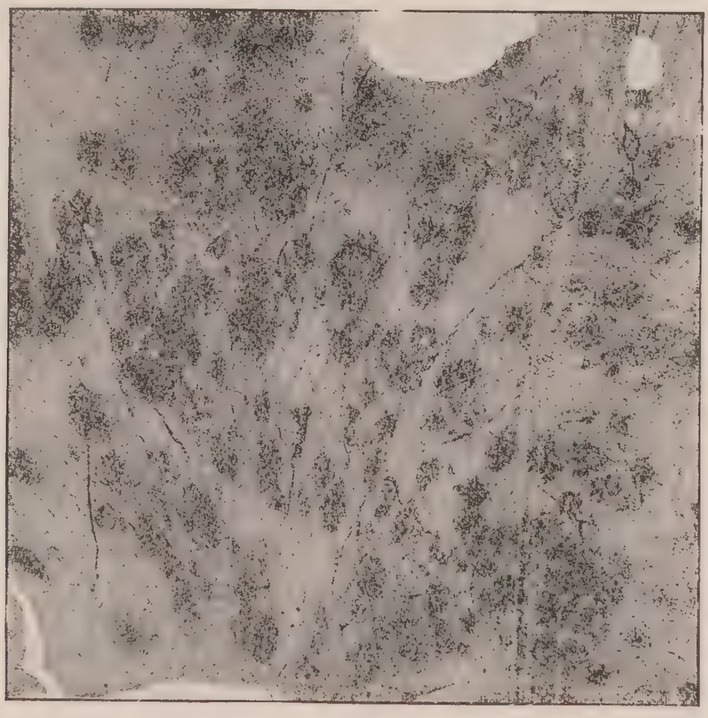


Fig. IX.—Photo of sections of the brain from case 2, Nickel Carbonyl Works. Showing hæmorrhages in the white matter, especially of the corpus callosum and internal capsule. (Mott).

The pons and medulla exhibited like vascular changes, most marked in the pyramids, and in a few places hæmorrhages were visible to the naked eye in the floor of the fourth ventricle. In the spinal cord there

were hæmorrhages in both the grey and white matter in the whole of the cervical and two or three upper dorsal segments, and in some places the anterior horns appeared to be damaged, particularly about the third and fourth cervical segments. The membranes were not thickened.

Microscopic Appearances.—Two methods of examination were employed. Duplicate portions of brain were taken from different regions, viz.:—cortex, corpus callosum, corona radiata, cerebellum, pons, and medulla, four blocks at different levels, and from the spinal cord, also four blocks from different levels. One set of specimens was treated with alcohol and embedded in paraffin, the specimens thereafter



Nickel Carbonyl Poisoning. Case 2.
Fig. X.—Shows a section of the corpus callosum of the brain stained with methyl blue and eosin, with hæmorrhages. ×10. (Mott).

sectioned and stained with Nissl stain or a modification. The duplicate specimens were placed in Müller's fluid and, after two weeks, changed into Marchi's fluid, the reaction from the latter being expedited by conducting the operation in a warm chamber at 37°C. for a week. Sections were thereafter cut in celloidin.

Most of the cells of the cerebral cortex showed diffuse staining, and the practical absence of granules in the cell-body and processes. The same conditions were observed in the cells of the pons and medulla. The cells of the various cranial nuclei of the medulla exhibited the following conditions in detail:—there was no change visible either in the small or large cells of the hypoglossal or facial nucleus, the cells of the respiratory

nucleus of Bechterew were found changed in all the sections, the protoplasm of cells and processes showing no granules and only diffuse staining; and the same observation applies, but in a lesser degree, to the cells of the nucleus ambiguus and the nucleus lateralis.

In the spinal cord in some places the anterior horn cells were found to be normal, but alongside these were cells profoundly changed. The vascular changes in the grey and white matter may be summed up as follows:—Naked-eye examination showed the white matter to have the largest number of hæmorrhages, which were most marked where there were terminal arteries. These hæmorrhages were not all of the same age, some being quite recent and others much older. These hæmorrhagic

Nickel Carbonyl Poisoning. Case 2.

Fig. XI.—A section of the medulla oblongata showing the condition of the cells in Bechterew's nucleus. The cells are diffusely stained throughout, and there is a proliferation of young neuroglia cells in the neighbourhood. ×350. (Mott).

areas revealed:—(1) A central area of degenerated nerve fibres, surrounded by a large number of black-stained granules. These were old foci. When stained by the Marchi method, the central portion and the leucocytes showed a large number of black-stained granules. (2) Foci of capillary hæmorrhages of more recent date, in which there was a central area of degenerated fibres staining a dirty pink by methylene blue and raffnain, surrounded by leucocytes, lymphocytes, and red blood corpuscles. (3) Foci of quite recent capillary hæmorrhages, in which extravasation only of red blood corpuscles into the tissues was found, there being few leucocytes and no glia cell proliferation. Very rarely were there black particles observable by the Marchi staining.

The vessels themselves appeared for the most part to be in a state of acute inflammation, as both arteries and veins in the grey and white matter showed leucocytes in the perivascular lymph sheaths in great abundance, and the capillaries almost everywhere a proliferation of nuclei.

In the nerve tissue itself there was a great excess of leucocytes and glia nuclei. In the medulla, about the level of the top of the calamus,

Fig. XII.—Section of brain tissue showing recent capillary hæmorrhage into white matter of the brain. Degenerated fibres stained black by Marchi method of staining. (Mott).

an artery, in a series of sections, was found to be filled with fibrin and leucocytes, evidence of thrombosis. Examination by the Marchi method, the sections being cut in celloidin, gave marked results of toxic action, as every part of the nervous system examined showed the presence of fibres, which had either become or were in the process of becoming

degenerated. These fibres, however, were not most numerous where the hæmorrhages were most frequent, as, for example in the corpus callosum, probably because the hæmorrhages in this region were the most recent to occur, and sufficient time had not elapsed to enable the process of degeneration to develop. Mott is of opinion that it requires six days after a hæmorrhage before the evidence of a degeneration becomes very obvious. Hence, he concludes, in this case, owing to the manifest and extensive degeneration which he found in the white substance of the cerebral cortex, of the cerebellum, the corona radiata, the pyramids, and the antero-lateral and posterior columns of the spinal

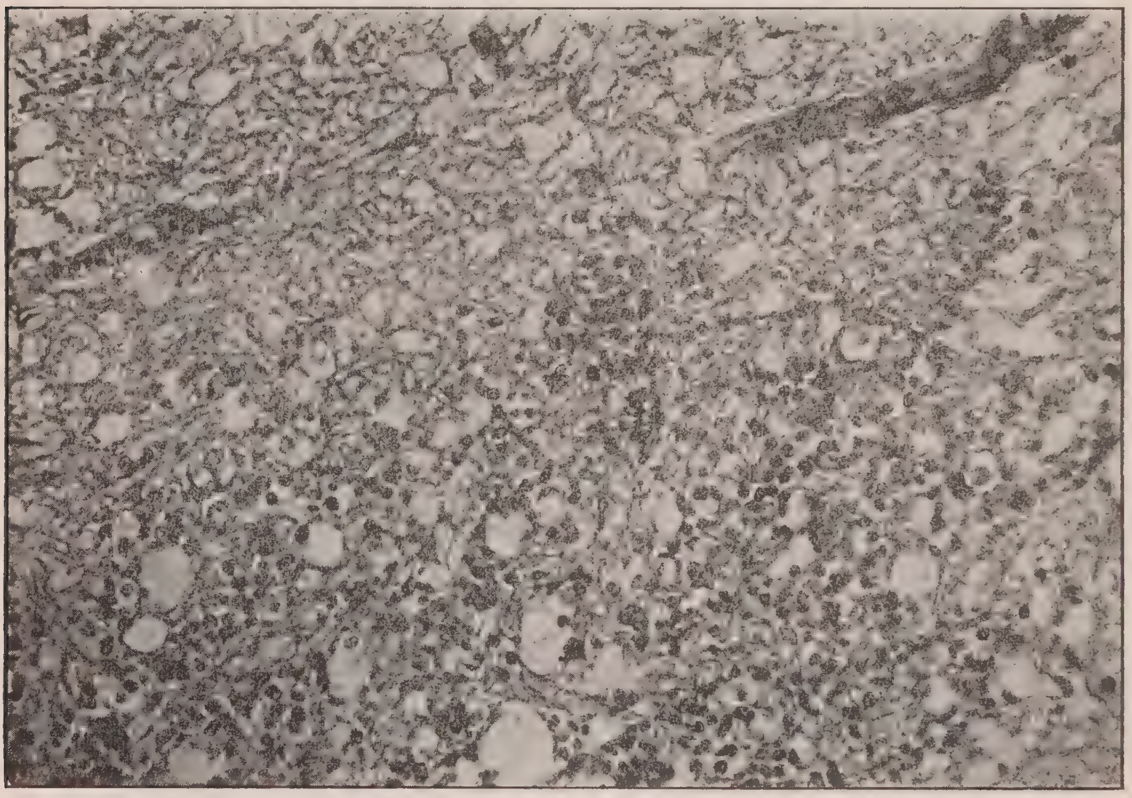


Fig. XIII.—Showing recent capillary hæmorrhage in the white matter of the internal capsule of the brain. ×280. (Mott).

cord, that these changes were associated with older foci of hæmorrhage. He also notes that what is observed is not a true degeneration process, because the degenerated fibres can be seen in their long axis.

From these findings, Mott is convinced that the profound changes observed in the central nervous system may be associated with (1) the poisonous gas, and (2) the clinical symptoms manifested by the patient during the attack; that the changes in the cardiac and respiratory nuclei of the medulla may possibly be associated with the rapid respiration and accelerated cardiac action, although it is not improbable that fatty degeneration of the heart and the hypostatic pneumonia may have con-

tributed; that the increase of the deep reflexes was due to degeneration of the pyramidal tract systems; and that the onset of delirium, ending in coma, may be associated with the recent extensive hæmorrhages found throughout the white matter of the brain, and with the marked changes in the cerebral cortex.

It is not unimportant to note in these two fatal cases of poisoning by the inhalation of the fumes from nickel carbonyl, that pneumonia developed in each patient during the currency of the seizure, an occurrence by no means uncommon in inhalation of CO gas from other and more common sources.

Mott has further extended his observations of the effects of this nickel compound, by examining the brains of animals which had died from exposure to the fumes from it, and, in addition, the brains of animals poisoned by coal-gas, and with carbon monoxide itself. These animals generally died within thirty-six hours after the first exposure, but he did not find any hæmorrhages visible to the naked eye in the cerebral white matter of the animals as he had found in human beings who had died from inhalation of coal-gas and nickel carbonyl. Microscopic hæmorrhages, however, were found, similar to those in human beings, in the pons and medulla, as were also chromolytic changes in the ganglion cells in proximity to these hæmorrhages.

In the brain of a rabbit, for example, which survived three days after exposure to the vapour of nickel carbonyl, engorgement of blood vessels and hæmorrhages, especially in the medulla oblongata, commencing endothelial mitosis, and glia cell proliferation, were exhibited as in the human cases. When stained with polychrome and eosin, the blood corpuscles mainly took the basophil stain and appeared blue, the surrounding serum being pink in colour. This was also similar to the behaviour of the blood to the same stain in the human cases. He endeavoured to reproduce this reaction by passing coal-gas through normal blood, but failed to get it. This, in his opinion, supports the view of Lancereaux that the protoplasm of the corpuscles in fatal cases of CO poisoning undergoes a change. Lancereaux had demonstrated that when a bat was subjected to the influence of this gas, an accumulation of blood formed in the small blood vessels of the transparent web or wing of the animal, and he formed the opinion that the red blood corpuscles perished under the influence of the gas, and that they gradually blocked the vessels and led to thrombus formation. This view, it may be noted, is closely akin to that of Poelchen, who arrived at the conclusion that the gas produced a disease of the brain blood-vessels, and a consequent necrobiotic change in the brain-tissue supplied by these vessels.

Effects on Nervous System of Persons Recovering from Acute Poisoning.

In previous chapters, considerable attention has been given to the effects upon the nervous system of those who have recovered from an attack of poisoning by carbon monoxide gas. Symptoms of the effects have been dealt with at some length, most of which have their origin in the lesions produced, temporarily or permanently, by the action of the gas on the central nervous system.

Of the graver lesions which have followed, mention may be made of multiple sclerosis and of disseminated myelitis.

The case recorded by Becker following exposure to illuminant-gas which is recorded already (p. 224) may be recalled. The symptoms recorded as indicative of multiple sclerosis may be summed as follows:—tremor, scanning speech, intentional tremors, shaking of the hand when writing, staggering gait and giddiness. These showed themselves progressively during the early months after the exposure, and at the end of five years had become gradually worse. The case cited by Avramoff (p. 225), after exposure to the fumes of burning charcoal, showed a like line of symptoms, which existed three years after exposure.

What has been designated as disseminated myelitis or acute encephalo-myelitis by Panski in his record of the symptoms of the case recorded by him is, perhaps, an isolated example, but the symptoms indicate a profound change in the central nervous system, viz., spastic, not degenerative, paralysis of the lower limbs, incontinence of urine and fæces, vaso-motor and trophic disturbances, somnolence lasting for days, dulled cerebration, slow, indistinct speech, and loss of memory (p. 235).

Of motor disorders met with, mention may be made of paresis, paralysis, paraplegia, hemiplegia, and monoplegia.

Paresis generally disappears, however, after some hours, but occasionally it may last for several weeks or longer, being usually accompanied by muscular inco-ordination. Motet's personal experiences are worth noting in this connection (p. 229), as also Ross and Bury's case (p. 231).

Perhaps the most noteworthy feature of paralysis after poisoning by this gas is its variability as to type and seat.

Of the sensory disturbances the most common manifestation is anæsthesia. Of this little, however, needs now be added. Reference to cases will demonstrate its variability as to extent and character.

But the trophic and vaso-motor disturbances deserve some attention. Ranging in certain cases from simple areas of erythema, with or without herpes, to deep sloughing of tissues, even to a degree involving exposure of muscles and opening of joints, they show that the effects of the gas on the nervous system are more profound than at first might be supposed likely.

Optical disturbances, including not merely paralysis of the ocular muscles, but involving the optic nerve structure producing blindness, have on a few occasions been recorded. In the case recorded by Sibelius (p. 267) in which the patient became completely blind in both eyes, followed by weakness of the extremities, epileptiform seizures, dementia, and death three months after exposure to the gas, serious progressive degenerative changes are indicated, as the *post-mortem* examination revealed softening of the cortex of the brain in both occipital lobes, involving nearly completely the calcarine fissure. It may be said shortly, that when ocular mischiefs follow exposure to the gas, these are usually accompanied or shortly afterwards followed by serious mental disturbances.

With regard to affections of speech, it may be said that when a person has been unconscious for some time from the effects of this gas, speech is commonly found to be more or less affected, and for variable times of duration. The character of the speech is blurred and indistinct, with gulping articulation. Sometimes syllables are lopped from words, sentences are uncompleted, and there is a tendency to repeat words or phrases. It is only, however, on comparatively rare occasions that the condition assumes the serious character of aphasia, but in the case recorded by Poelchen (p. 272) mental symptoms with aphasia developed at the end of three weeks after exposure. The case recorded by one of us presents interesting details relative to aphasia (p. 295).

Mental Disorders.—There cannot be the least doubt that, in serious cases of poisoning, mental disorders of a grave type are apt to develop. These are usually progressively developed in the train of a line of symptoms showing grave interference with the action of the central nervous system.

The pathological lesions found after death from acute poisoning in the brain and spinal cord readily indicate whence the origin of these manifestations. There can hardly be reasonable doubt that these lesions are the result of the multiple small hæmorrhages into the nervous matter of the brain and cord, but, perhaps mainly, to the condition of the blood vessels which is found to follow in their train, viz. :—thickening of the walls of the cerebral and spinal vessels, contraction of their lumen, and to consequent decreased blood-supply, thus leading to degeneration and softening. It would only be multiplying words to elaborate this.

CHAPTER XV.

METHODS OF DETECTING CARBON MONOXIDE IN THE BODY AND IN AIR OR GASES.

There are many circumstances in which it is necessary to be able to say that carbon monoxide has been detected in the air in which a person has been found dead, or in the body of that person. While the circumstances may warrant an assumption that the cause of death has likely been this gas, it is always more satisfactory for legal purposes that the gas should be looked for, either in the air or in the blood of the person exposed to the suspected air. Particularly is it desirable in those cases in which persons have been found unconscious in circumstances in which it is not unreasonable to suppose that the state of the person has been due to a gaseous cause, and especially in cases in which actions for compensation may arise.

Our experience hitherto has been that this has been too much neglected, and that dependence for the opinion offered as to the cause has been solely placed upon the clinical symptoms. Moreover, when the need for such an examination of the blood has arisen, too long time has been allowed to elapse to secure the necessary evidence. We are strongly of opinion that medical men who have to deal with workers in mines and other occupations in which exposures to carbon monoxide are incidental to the employment, and especially where employees are found unconscious or dead, should as a matter of routine make an examination of the blood for the spectrum of this gas. Short of this, the evidence is not so certain, although there are more simple chemical tests by which the presence of the gas in the blood in severe or fatal cases may be determined with reasonable accuracy. It is, however, necessary to point out at the outset, that there are no simple methods of determining small percentage amounts of this gas, either chemically or by chemical tests, the only available and certain method in such cases being the discovery of the spectrum by the micro-spectroscope.

It is not to be expected that the average medical man should be familiar with the practical conduct of quantitative chemical tests for this gas in air, but he ought to be able to apply the more simple chemical tests, and make the necessary spectroscopic examination.

Examination of the Blood.

In the case of persons who have been severely poisoned by the gas itself, or by a compound gas of which carbon monoxide is a known constituent, and in cases where exposure to atmospheres containing it has resulted fatally, the blood is more or less saturated with it, and CO-hæmoglobin will be found in greater or lesser amount therein; consequently the blood generally will be found to be of a more or less arterial red or cherry-red colour. If, therefore, in such cases one cubic centimetre of blood be mixed with recently-boiled and cooled potable water so as to form a total mixture of 100 c.c., the resulting colour will be found to be more or less of a rose-pink colour. In order to compare this with normal blood similarly treated, like amounts of normal blood and water respectively are used, and the mixture compared in colour with that from the body of the patient or the deceased person. The contrast will be definite in character, because the addition of water to normal blood causes the mixture to assume a lake or light-yellow colour, whereas the blood containing the CO-hæmoglobin will be more or less rose-pink in colour. This contrast is likely to be found in all severe cases of poisoning, no matter whether the source of the gas has been from a lime-kiln, air of mines, illuminating-gas, coke or charcoal stoves, or gas-geysers.

It is not necessary to discuss further the percentage amounts of carbon monoxide present in the air of mines under certain circumstances, since these have been alluded to several times in preceding pages. While deaths from exposure to the gases from lime-burning operations are, perhaps, not rare, it is seldom that the amount or degree of saturation of the blood with carbon monoxide has been estimated from examination of the blood of those who have succumbed. One of us[1] had an opportunity of making an examination of the blood of a woman who, along with her two children aged 4 years and 1½ years respectively, died in her house, which was in close proximity to lime-kilns, from the gas which had passed through the defective gable of the house into the apartment which she and her children occupied. The degree of saturation of the blood of the woman was 70 per cent.

Deaths from exposure to illuminating-gas, due either to accident or suicide, are, however, much more common. In the communication, above alluded to, several cases are recorded of accidental deaths from this cause. In one of these, no fewer than seven persons, composing a family of father, mother, and five children of varying ages, died, although at the time of discovery the father was found alive but deeply unconscious. In more recent cases which one of us has seen, one or more

[1] Glaister: *The Lancet*, 8th and 15th December, 1906.

persons, including animals, have succumbed to the influence of the gas. In all of these cases, the source of the gas has been underground leaks in main or sub-main pipes, since no gas-pipes had been laid into the houses in question. One of us (Glaister) has seen several cases of suicide by illuminating-gas. In some of these the gas-cock in the occupied room had simply been turned on full, and in others the gas had been led to the air-passages of the individual by a rubber-pipe from the gas-supply. In a recent case in which suicide was effected in this manner, and where the arrangements referred to were found on discovery of the body of the deceased woman, the degree of saturation of the blood with CO-hæmoglobin was 34 per cent. In another case, the individual tried to commit suicide by placing his head within a cooking-oven supplied with gas. He was found, however, before death had supervened, but he was unconscious. He eventually recovered under active treatment.

The amount or degree of saturation of the blood with CO-hæmoglobin in such cases depends upon (1) the character and composition of the illuminating-gas; and (2) the length of time of exposure before discovery. With regard to the former condition, it will especially depend upon the extent to which carbon monoxide is present how soon unconsciousness and death will be likely to supervene.

Coal-gas, when not admixed with water-gas or carburetted water-gas, contains on the average from 6 to 10 per cent. of carbon monoxide, whereas producer or water-gas contains as much as from 25 to 50 per cent., and carburetted water-gas about 30 per cent. In England and Wales, for a population equal in number to the population of London in 1896, the number of accidental cases of gas-poisoning in places where coal-gas alone was used, was three; in places outside London, where the same condition obtained, the total deaths numbered 4·2; and in places outside London where coal-gas partly mixed with water-gas was employed, the total deaths numbered 10·4. From the Board of Trade Returns of Gas Undertakings in Great Britain for 1904, it will be found that of 454 undertakings other than of local authorities in England and Wales, 68 or nearly 15 per cent. of the total producers of this class, supplied gas for illuminating purposes which contained some measure of admixture with water-gas; that in Ireland of ten undertakings of this class, three supplied some admixture; and that in Scotland, none supplied any admixture or proportion of carburetted-gas. From the Returns for 1905, of 208 municipal or public local authorities who were gas undertakers in England and Wales, 28 or 13·5 per cent. supplied admixtures; of 46 in Scotland, 3 or 6·5 per cent., did the same; and in Ireland, of 11 municipal producers, only 1, or 9 per cent., provided admixture of carburetted-gas with coal-gas.

The Returns for that year show that the percentage amounts of carburetted gas in the admixtures actually purveyed by gas undertakers other than local authorities in England and Wales varied considerably. These ranged, indeed, from 3 per cent. in Waltham up to 60·7 per cent. in Southgate. Southend, Liverpool, North Middlesex, Tottenham, Norwich, Bath, and Dorking supplied gas containing between 50 and 60 per cent. of carburetted-gas; Maidenhead, Lea Bridge, Folkestone, Wandsworth, Brentford, Harrow, Hornsey, Croydon, Bexhill, Hastings, and Horsham, between 40 and 50 per cent.; Falmouth, Bournemouth, Gosport, Watford, Gravesend, Preston, Stretford, London (Commercial and Suburban), Hampton Court, Taunton, Epsom, Wimbledon, Redhill, Brighton, Bridlington, and Hull Station, between 30 and 40 per cent.; while Durham, Hartlepool, Colchester, Ilford, Romford, West Ham, Swansea, Dartford, Chatham, Tonbridge, Ramsbottom, Staines, Newport (Mon.), Rushden, Newcastle, Caterham, Guildford, Kingston-on-Thames, Eastbourne, Malton, Scarborough, and York only between 20 and 30 per cent. The remainder of such gas undertakers supply less than 20 per cent. In Scotland, as has been noted, no gas company in this class purveys carburetted-gas. In Ireland the percentages run from 47 per cent. in Dublin, to 20 per cent. in Londonderry and Wexford.

The percentage amounts actually supplied by municipal or other local authorities who are gas undertakers in England and Wales varied from 12 per cent. in Leicester to 55·86 in Southport. Oldham supplied between 40 and 50 per cent.; Burnley, Manchester, Lincoln, Longton, and Coventry, between 30 and 40 per cent.; Stockport, Carlisle, Stockton-on-Tees, Accrington, Blackburn, Chorley, Todmorden, Loughborough, Stafford, Birmingham, Oldbury, Halifax, Hebden Bridge, and Leeds between 20 and 30 per cent.; and the remainder less than 20 per cent.

Of the three municipal undertakers in Scotland, Dundee added 25 per cent., Edinburgh 4.23 per cent., and Paisley less than 2 per cent. of carburetted gas.

In the twelfth Annual Report of the Gas and Electric Lighting Commissioners of the Commonwealth of Massachusetts, 1896, the Commissioners furnish information respecting the cases reported of death and bodily injuries from gas-poisoning during that year. They state that the attention of the Board had been directed to seventy-six different cases of escaping gas, from which the deaths of 51 persons and the bodily injury of 118 persons had resulted. In the preceding years, 1893, 1894, and 1895 in the same State, the deaths from the same cause numbered, 23, 23, and 22 respectively. In the thirteenth Report, 1897, 60 persons died and 74 others were injured by gas-poisoning. In Boston alone 75 per cent. of these fatal cases occurred. It is noteworthy to observe that in some

of the instances in which a fatal issue ensued, conditions of comparatively free ventilation existed in the apartments in which the deaths took place.

In other cities of the United States of America, in which admixtures of carburetted-gas have been adopted, similar increases in the death-toll have been recorded. In the above-named Reports, as also in that of the Sanitary State Board of Health of Massachusetts, it is stated that in the combined populations of New York, Brooklyn, and Baltimore, for the thirteen years prior to the introduction of water-gas into the supply for illuminating purposes in 1880, there had only been registered 16 cases of gas-poisoning, whereas during the seven and a half years after its introduction the number of deaths from that cause had risen to 120, of which, in some of the latter years of that period, New York alone had contributed in an individual year as many as 30 deaths. According to Bayley,[1] the gas supplied by the companies for illuminating purposes to all the cities of New York State consisted of carburetted water-gas containing from 30 to 45 per cent. of carbon monoxide.

Cattel writes of Philadelphia that "there seems to have been recently an epidemic of accidental and suicidal deaths from illuminating-gas. During 1896, the coroner held inquests on 33 cases in which death was determined to have been directly due to this cause. . . ."[2]

Water-gas is largely used in Philadelphia, the CO forming one-third of the illuminating-gas. In 1897, the deaths in that city numbered 34 from this cause, of which 14 were accidental, 18 were suicidal, and 2 were homicidal.

In twenty-two other cities and towns of the United States, the deaths in 1889 from gas inhalation numbered 107, in 1890, they rose to 174, of which 139 were accidental, and in the first half of 1891 they numbered 100.

The findings of the Committee on Water-Gas appointed by the Local Government Board for England in 1898, to the effect that the adoption of carburetted water-gas by the gas companies of this country created an increased risk of danger to health and life, because of the increased content of carbon monoxide, seem not only to be warranted by the facts but form a self-evident proposition.

In view of that general finding, the Committee in their Report submitted the following recommendations, viz :—[3]

(1) That it should be illegal for any person to make and distribute for heating and lighting purposes, any poisonous gas which does not possess a distinct and pungent smell. (2) That all persons applying for

[1] *Medical News of New York*, 23rd August, 1902; *Lancet*, Vol. II., 1902, p. 1007.
[2] *International Medical Magazine*, 1897.
[3] *Report of the Departmental Committee on Water-Gas*, 1899.

statutory powers to make and distribute gas, should be required to state in their application the kind of gas which they propose to sell, viz.: whether ordinary coal-gas, carburetted water-gas, plain water-gas, or other variety of gas—separately or mixed. (3) That before any kind of water-gas is distributed in any place due public notice of the proposal should be required to be given, and that so long as there is any water-gas in a gas-supply that fact should be stated on every demand note. (4) That where water-gas is distributed records should be kept by the producer showing the respective amounts of the gases issued day by day, distinguishing the gas supplied to each area (if more than one and separately served) and the day and night supply; that these records should at all times be open to inspection by any gas consumer or ratepayer of the district and should be published quarterly in a local newspaper; and that a new column should be added to the annual returns made by the Board of Trade giving the total amount of water-gas issued as compared with coal-gas. (5) That power be conferred on a central department to make regulations enforceable by adequate penalties, limiting the proportion of carbonic oxide in the public gas-supply to 12 per cent., or such greater amount as the department may consider desirable. These regulations might be applicable either generally over the United Kingdom or to any particular locality, and might contain such conditions, if any, as appeared necessary. (6) That powers should also be given for the regulation of the distribution and use of gas by means of bye-laws, made subject to the approval of a central department and administered under local control. The matters to be so dealt with might include the following:—the hours during which or the arrangements by which the limit imposed on carbonic oxide should be enforced; the use in emergencies of more than the authorised proportion of carbonic oxide; the character of the gas-burners, fittings, and apparatus to be used, having regard to the circumstances of their employment; the testing of the gas; and other similar questions. (7) That the provisions of sections 28 to 34 of the Gasworks Clauses Act, 1871, should be made applicable to the testing of gas for carbonic oxide, and that in all cases where a limit has been placed on the carbonic oxide in the gas-supply of a locality there should be some person empowered and required to test for carbonic oxide and to publish the results periodically. (8) That some steps should be taken to secure that the verdicts of Coroners' juries should lend themselves more readily to clear classification by the Registrar-General, and in particular that in every case where the Coroner is in possession of a medical certificate of death or where medical evidence has been taken at the inquest, a copy of that certificate or an abstract of that evidence should be appended to the Coroner's certificate when sent to the Registrar-General.

So far as we are aware, the only outcome of the work of that

Committee and its recommendations has been the incorporation in the Board of Trade Returns of Gas Undertakings of information concerning the amounts of carburetted water-gas made and used by Gas Undertakers.

The effect of the inhalation of carbon monoxide from defective apparatus conveying illuminating-gas containing admixtures of carburetted water-gas has been to create an increased risk of danger to health and life, because of the increased content of that noxious constituent.

It will be obvious, therefore, from the foregoing, that the colour of the blood of a person after exposure to illuminating-gas will, perhaps, more largely depend upon the percentage proportion of carbon monoxide gas it contains than upon the time of exposure; in other words, that the degree of saturation of the blood with that gas will be more quickly attained with coal-gas, enriched with water-gas or carburetted water-gas, than with ordinary coal-gas not so admixed. Where, on the other hand, the mixture of gases is relatively rich in carbonic oxide, and especially where the air is mainly composed of that gas, the time of exposure becomes the more important consideration with respect to its lethal action.

It is for this reason that Haldane and others have suggested the use of small animals, such as birds or mice, as a test-medium of the presence in high amount of CO gas in the air of coal-pits. In respect that these small animals have a much more rapid respiration than man or the larger animals, an amount of carbonic oxide gas will act prejudicially more rapidly upon them than upon man, indeed, will operate harmfully before the gas has had time to act inimically upon man.

Colorimetric Test.

If we assume, then, that circumstances have arisen in which it is necessary and desirable to discover whether or not a living unconscious person is suffering from the poisonous action of carbon monoxide gas, or that a dead person has died from its effects, the simplest test of the presence of the gas in the blood—the union of the gas with the hæmoglobin—is the colorimetric test. That test ought to be conducted as follows:—

Take three test tubes, each of about three-eighths or thereby of an inch in diameter, and about four inches in length. Let them be marked No. 1, No. 2, and No. 3. Into No. 1 put one drop of the blood to be examined and add 99 drops of ordinary potable water.

Into No. 2 put one drop of ordinary blood obtainable from the finger of the operator and add the same amount of water.

If carbon monoxide gas be present even in small amount in No. 1, the colour resulting will be more or less pink in hue, which will be more

I.

II.

III.

I. Tint of Solution of one per cent. of Normal Blood in Water.
II. Do. do. of Blood containing some CO Gas.
III. Do. do. of Blood saturated with CO Gas.

noteworthy when compared with the colour resulting in No. 2, because being more or less yellow or lakey in hue.

In order to form some rough idea what amount or degree of saturation with carbon monoxide is present in the blood of the person under examination, it will now be necessary to prepare blood which is fully saturated with that gas. That is accomplished by putting some fresh blood into a stoppered glass bottle, and with the aid of a small rubber tube passing through it ordinary illuminating-gas from the nearest gas-jet, and repeatedly shaking the bottle on replacing the stopper. This operation is repeated sufficiently often until the colour of the blood in the bottle has attained its highest crimson-red or cherry-red colour.

At this stage add to test-tube No. 3 one drop of this blood diluted as the others, and compare the colour of tube No. 3 with that of tube No. 1. It will be found in every case that the blood of No. 3 will be more crimson in colour than No. 1, the difference depending upon the different amounts or degrees of saturation with CO in the blood in No. 1 and that in No. 3.

In order to be able to say that CO is present, it is enough to determine that the blood in No. 1 is crimson or pink in colour as compared with that in No. 2, the use of No. 3 being only to determine approximately the degree of saturation with the gas.

For accurate quantitative estimations of degrees of saturation, there is, perhaps, no more satisfactory method than that suggested by Haldane.[1] This, like the qualitative test, is a colorimetric test, and depends upon the colour of blood under inspection relative to that of an equal amount of blood fully saturated with CO gas, the matching of the former to the colour tint of the latter being effected by a standard solution of carmine.

The following is a summary of the technique of the method :—

(1) A one per cent. solution of the blood to be examined is put into a test tube;

(2) Into a like tube is put a solution of normal blood of the same strength;

(3) Into a third tube is put a solution of normal blood of the same strength which has been saturated with CO gas obtained by shaking it with ordinary illuminating-gas until its fullest pink colour has been attained. In order to ascertain the degree of saturation of the blood under examination, it is now necessary to match the tints in the tubes Nos. 1 and 3. In the bulk of cases, probably, it will be found that the tint in No. 1 is less crimson or pink than that in No. 3, although it will be crimson or pink in colour compared with the yellow colour in No. 2.

[1] *Jour. of Physiology*, Vol. XVIII., p. 430 *et. seq.*; *ibid.*, p. 463; *ibid.*, Vol. XX., p. 521; Vol. XVII., p. 233 and p. 478; *Trans. Inst. Min. Eng.*, Vol. II., p. 502; *ibid.* Vol., XXXVIII., p. 267.

To produce equality of tint in Nos. 1 and 3 tubes, a standard solution of carmine is carefully added to tube No. 1 until its colour is equal to that in No. 3. This is accomplished by adding the carmine solution drop by drop, with continual shaking of the tube, until the desired equality of tint is obtained.

The standard solution of carmine is made by mixing one gramme of pure carmine with a few drops of ammonia in a mortar and dissolving in 100 c.c. of pure glycerine, and the *using* standard solution is made from this by diluting 5 c.c. up to 500 c.c. with distilled water.

When about 6 c.c. of this using standard solution are added to 5 c.c. of ox blood so diluted with distilled water that the resulting solution contains one per cent. of blood, the colour or tint formed will be equal to that of blood similarly diluted but saturated with CO gas.

The procedure then is as follows :—The diluted blood under examination is first compared with that of similarly-diluted normal blood. If it be crimson or pink in colour as compared with the yellow colour of the normal blood solution, it may then be considered to contain some measure of CO gas in union with the hæmoglobin. This can now be confirmed with the spectroscope. If, on the other hand, the colours of both solutions are alike yellow in colour, it may be concluded that there is no CO gas present in the blood under examination.

Assuming, however, that this blood is decidedly more crimson or pinker in colour than normal blood, it is now necessary to ascertain the amount of CO present. It should now be compared and contrasted with the blood artificially saturated with that gas, when it will probably be found to be less crimson or pink in colour tone than the latter.

At this stage, the using carmine solution having been put into a burette, the solution should be added drop by drop, with continuous shaking of the tube contents, to the blood under examinaton, until by repeated comparison the tints are exactly matched.

From the amount, of carmine solution added, the degree of saturation of the blood-sample under inspection may now be calculated.

If, for example, 1·5 c.c. of carmine solution are needed to match the colour, and on the assumption that exactly 6 c.c. of the carmine solution are needed to match the tint of a 1 per cent. solution of normal blood with the similar dilution of artificially CO-saturated blood, the calculation will be as follows :—.

$$\frac{100 \times 4{\cdot}5 \text{ (that is: } 6 - 1{\cdot}5)}{6} = 75 \text{ per cent.}$$

in other words, the hæmoglobin in the sample under inspection has taken up CO gas to the extent of 25 per cent. of complete saturation ; that is, $100 - 75 = 25$ per cent.

The calculation may be checked in the following manner :—Instead

of adding the carmine solution to the blood under examination and comparing it with the artificially-saturated blood solution, the solution of carmine may be added to the dilution of normal blood till its tint exactly matches with the tint of the blood under inspection. If the former estimation be correct, it will be found that 1·5 c.c. of carmine solution have been required to match the tints, thus :—

$$\frac{100 \times 1{\cdot}5}{6} = 25 \text{ per cent. of saturation ;}$$

which is the degree of saturation with CO gas of the blood under examination.

In this colorimetric test it is important to ascertain (1) what number of c.c. of the standard carmine solution employed are exactly needed to match the tint of blood fully saturated with the gas, and (2) to make parallel in all details the conditions of comparison, in order that accurate results be obtained.

Having used the foregoing test on many occasions, after testing the conditions upon which it is based, we can speak with some confidence of its value.

Spectroscopic Test.

The examination of suspected blood for the spectrum of CO-hæmoglobin ought to be made at some stage of the investigation. We prefer to make this the first test in ordinary course. The apparatus whereby to carry it out is, perhaps, familiar to most medical men, but we make no apology for introducing a description of it here. It consists of (1) a hand direct spectroscope which gives a good spectrum, (2) a holder on which it may be clamped at a suitable level, (3) a flat cell of oblong shape capable of holding about 5 c.c. of the diluted blood, and (4) a similar holder and clamp. The only chemical solution required is ammonium sulphide, preferably fresh, since then only is it likely to be free from deposited sulphur.

The strength of blood to be used for examination and to be placed in the cell ought not to exceed two per cent., but we prefer to use one per cent. It is a good plan to employ water which has been distilled and placed after having been boiled into small bottles filled to the brim. This is to prevent oxygenation of the diluted blood which would happen in some measure if ordinary water, which is aerated as it comes from a tap, were employed. When the amount of blood available is only that contained in a capillary tube, recourse must be made to the micro-spectroscope for the examination, as the amount of blood is quite insufficient for examination by the direct vision spectroscope.[1]

Carbon monoxide forms, as has already been said, a stable compound

[1] Glaister: *Textbook of Medical Jurisprudence*, 2nd ed., p. 347.

with the hæmoglobin of the blood which persists for many hours at least after death, and which is called carboxy-hæmoglobin or carbonic oxide hæmoglobin. The spectrum which it gives consists of two bands between the Frauenhofer lines D and E, or in other words in the yellow half of the green. This spectrum is closely alike to that of oxy-hæmoglobin or that of arterial blood, the main differences between them, short of counting the wave lengths of each respectively, being that in the latter both bands are nearer the violet end of the spectrum than in the former. For the less experienced observer this difference does not matter so much,

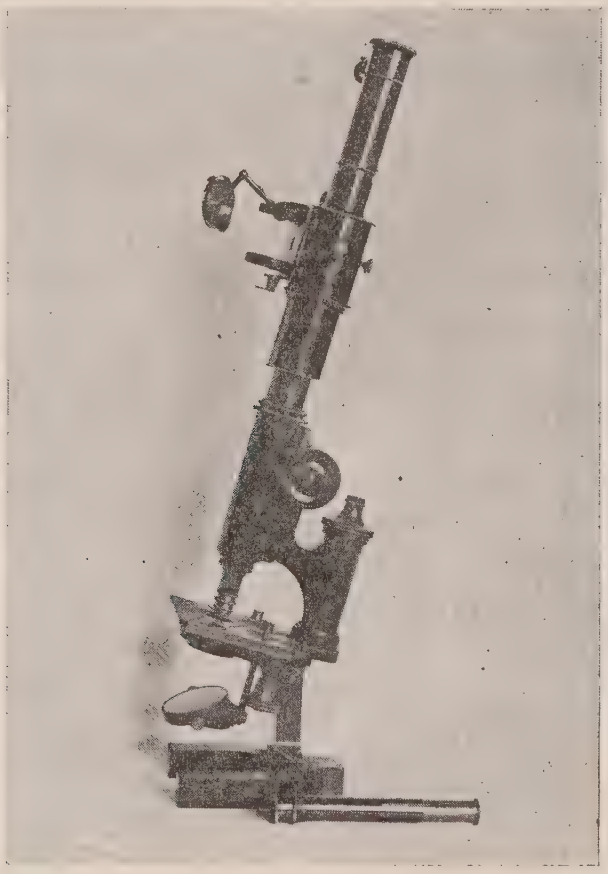
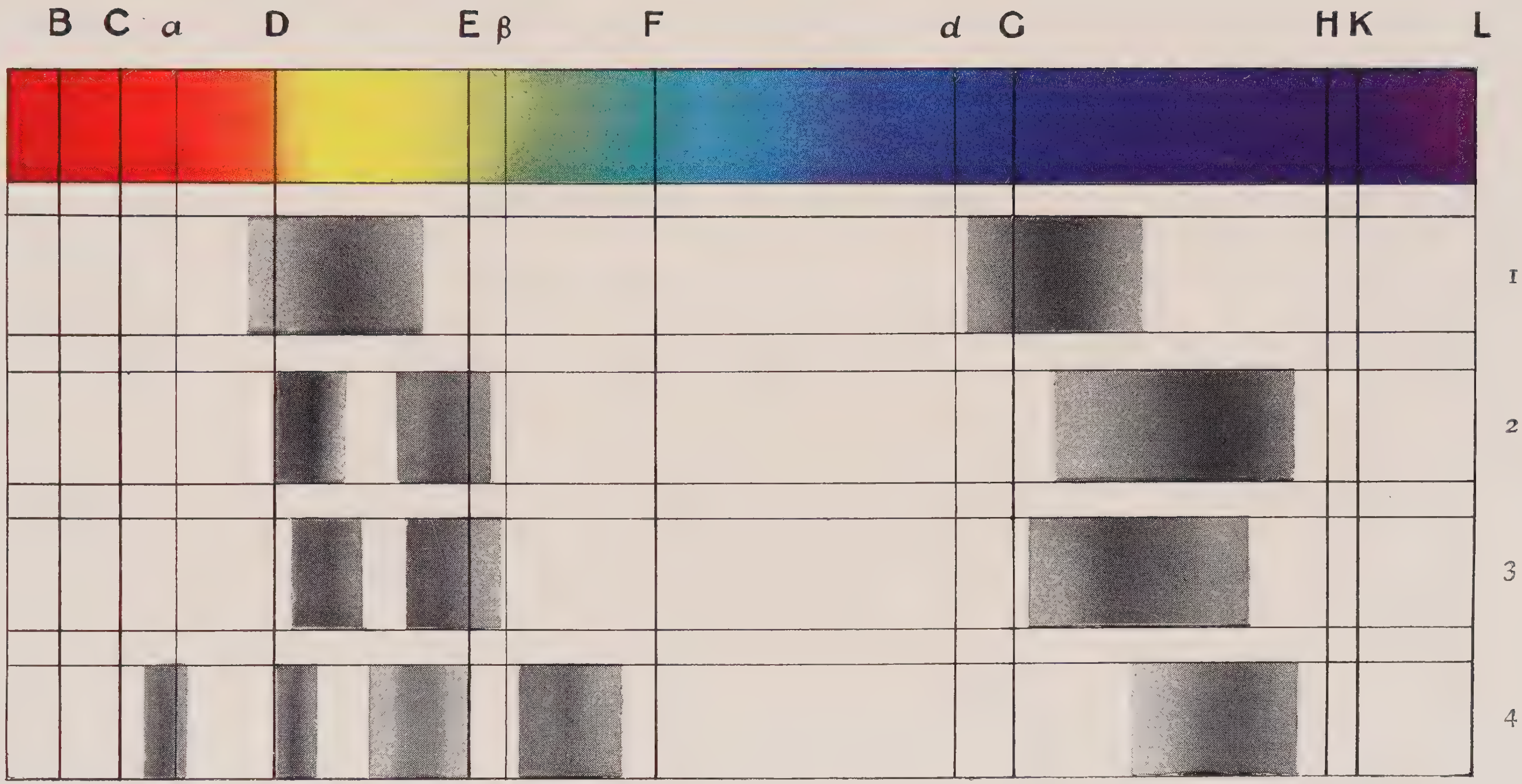


Fig. XIV.—Shows the microspectroscope fitted in microscope ready for use. Lying in front of it is a small hand direct-vision spectroscope.

since the spectrum of oxy-hæmoglobin is completely changed by the addition to the diluted blood of a drop of ammonium sulphide, whereas that containing carboxy-hæmoglobin is not so altered; to put it in other words, the spectrum of oxy-hæmoglobin is reduced by the addition of ammonium sulphide to the spectrum of *reduced hæmoglobin*, whereas that of carboxy-hæmoglobin is unchanged by the addition.

In all deaths by carbon monoxide, no matter what its vehicle, be it coal-gas, water, or producer-gas, or the fumes of lime-burning, or those from blaise heaps, the foregoing characteristic spectrum will be found.



1 Reduced Hæmoglobin. 2 Oxy-hæmoglobin. 3 Carboxy-hæmoglobin. 4 Methæmoglobin (neutral).

But as the physical appearance of blood varies as is the percentage amount of CO which has combined with the hæmoglobin, in like manner will the density of the spectrum vary. In some cases, indeed, there may be found, along with the two bands of CO-hæmoglobin, an additional band due either to some reduced hæmoglobin or to methæmoglobin. This has been found in certain cases. But it may be taken as the expression of a general rule that where CO has been a definite factor in causing death, the characteristic spectrum will be fonnd.

For the beginner or one called upon infrequently to distinguish the gas in the blood, it is a good plan to mix ordinary blood with water in the same dilution as that of the suspected blood, to subject each in turn to spectroscopic examination, to add to each a drop of ammonium sulphide, and again to examine spectroscopically, in order to detect the difference in the resulting spectra.

Certain **chemical tests** of a simple character have been proposed to detect the gas in the blood, but they do not compare in absolute reliability with the spectroscope. Of these mention may be made of (1) *Hoppe-Seyler's test*, which consists in adding a solution of sodium hydrate of 1.34 sp. gr. to the blood. If CO be present, a bright-red colour developes, but if the blood be normal, a dirty-green colour is the only result; (2) *Kunkel's test* consists in adding to the blood diluted with nine parts of water, a few drops of a 3 per cent. aqueous solution of tannin. In the presence of CO a pinkish-white precipitate will form, but if blood be normal, a brownish-white coloured precipitate.

Detection of Carbon Monoxide in Air.

It may be said once and for all that simple methods of determining this gas in air have not yet been devised, nor is it intended to discuss at very great length the many chemical methods, all more or less elaborate, which have been proposed. At the same time there are circumstances, such as the analysis of the air of mines and other places, which demand a reliable method of estimating quantitatively this gas as well as other constituents. To some of these allusion will be made. Meantime, there is one method connected with the blood reaction with the gas already dealt with, to which some attention ought to be given, and, moreover, which is capable of detecting small amounts of this gas in air.

Vogel's Method.

This is based upon the known formation of CO-hæmoglobin. The description will best be followed by the figure of the apparatus, depicted. The apparatus is prepared for the test in the following manner. Close the

constriction at *d* with a plug of glass-wool; fill the tube from *d* to *f* with glass powdered as fine as ordinary gunpowder; after sifting, digesting with HCl, washing with water, and drying it, moisten it *in situ* with distilled water; apply a water-pump connection at *e* to drain through the surplus water, which is run off by the stopper *c*; thereafter drop over the glass powder 2 c.c. of dilute blood (prepared as described below) the amount of dilution being 5 per cent.; gently blow through *g* to distribute the blood solution evenly through the glass powder; attach an aspirating vessel of known capacity at *e*, and aspirate through the apparatus about 10 litres of the air under examination at the rate of about 1 litre per 20 or 25 minutes. When room-air is under examination, the air ought first to be passed through a glass cylinder containing pumice stone moistened with water, which may be attached at *g*. When the above quantity of

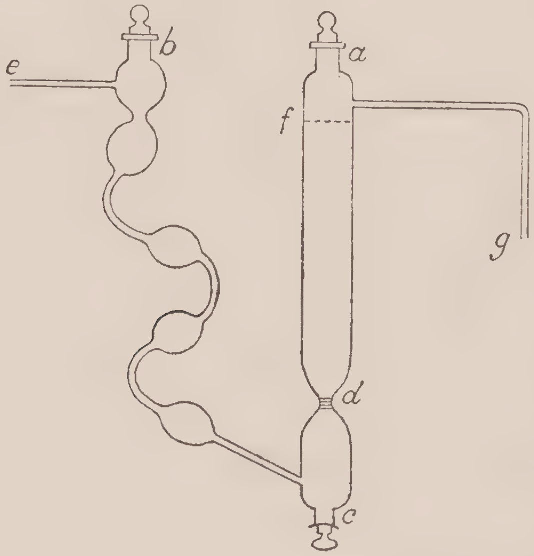


Fig. XV.—Vogel's apparatus for testing for carbon monoxide.

air has been passed, from 3 to 5 c.c. of distilled water should be added to the tube-contents at *a*, the aspirator disconnected, and the water-pump connected, so that the blood solution and the added water shall be drawn into the lower part of the tube and removed at *c* for spectroscopic examination. After the operation is over, the glass powder should be washed clean with water and drained, ready for another experiment.[1] (Fig. XV.).

The blood solution to be employed is obtained by mixing fibrin-free blood with an *equal volume of cold saturated solution of borax*, since by that addition decomposition is prevented, the spectroscopic properties are not interfered with, nor is the action with ammonium sulphide. One

[1] Glaister: *Textbook of Public Health*, 2nd ed., p. 155.

c.c. of this mixture added to 19 c.c. of distilled water is the strength to be used, and 2 c.c. is the quantity for each experiment.

The above test will detect 0·05 per cent. of carbon monoxide in air.

The blood is thereafter examined spectroscopically by the direct vision spectroscope for the spectrum of CO-hæmoglobin.

Estimation of Carbon Monoxide quantitatively in the Air of Mines and Other Places.

Many chemical methods have been devised from time to time to determine the amount of this gas in the air of mines and other places.

Probably among the earliest of these was the method of Winkler.[1]

The principles of this method may be summarised as follows:—The suspected air is made to pass through a dilute solution of sodium cuprous chloride, after the addition of a few drops of a solution of sodium palladious chloride. If CO be present, a black precipitate will be formed. Instead, however, of sodium being employed, later modifications of the method adopt the use of ammonia in substitution for the sodium.

The absorbent used in the process, therefore, is an ammoniacal solution of cuprous chloride. This is prepared by dissolving 250 grammes of ammonium chloride in 750 c.c. of distilled water, and thereafter adding 200 grammes of cuprous chloride. This latter salt will only dissolve after thorough agitation of the vessel and contents. When made, this solution will keep for a long time, provided that it is kept in air-tight stoppered bottles furnished with rubber stoppers, and also if a spiral of clean copper wire reaching from top to bottom is placed in the bottle.

Before this solution may be used for gas analysis, however, an amount of strong ammonia, equal to one-third the volume of solution used, must be added.

This ammoniacal cuprous chloride solution, it must be remembered, absorbs oxygen and carbon dioxide gases as well as carbon monoxide; consequently in any examination of air for carbon monoxide, the oxygen and carbon dioxide must have been previously removed by other suitable absorbent solutions before the estimation of carbon monoxide is made. Usually when Hempel's burettes are employed, the oxygen is absorbed by a solution of pyrogallate of potash and the carbon dioxide by a solution of caustic potash.

If estimation of carbon monoxide is made in Hempel's burettes and if more than one estimation is to be made at one time, it is best to keep the ammoniacal cuprous solution in a compound burette in order to minimise the chances of absorption of oxygen and carbon dioxide.

[1] Winkler and Lunge: *Tech. Gas Analysis*, 2nd ed., p. 74; Lunge: *Tech. Methods of Chem. Analysis*, Vol. I., p. 889.

For accuracy, however, it is better to use a fresh solution of the absorbent at each estimation : that is, 150 c.c. of cuprous chloride solution to which have been added 50 c.c. of strong ammonia.

One cubic centimetre of this solution is capable of absorbing 16 c.c. of carbon monoxide gas.

Potain and Drouin[1] have suggested a method of quantitative estimation founded upon the foregoing.

Other methods have been proposed which are based upon the conversion of the CO present into CO_2.

Spitta[2] suggested a method based upon the estimation of the carbon dioxide produced from the combustion of the carbon monoxide by means of silver electrodes coated with palladium and heated to 150° – 160°C., and estimating the CO_2 gas which results by Pettenkofer's method.

Another method is founded upon the oxidation of CO by iodine pentoxide, the reaction which takes place being as follows :—

$$5\ CO + I_2 0_5 = 5\ CO_2 + I_2.$$

This was the plan suggested by De la Harte and Reverdine.[3] When the reaction is effected at a temperature between 150°C. and 160°C., the test may be made a quantitative test ; but if that temperature be exceeded, or if methane or hydrogen be present in the air under examination, the reaction will fail in its purpose. Even at the temperatures stated, however, it is to be noted that hydrocarbons of the ethylene and acetylene series are attacked. Fortunately these are absent in the air of mines generally. Besides, hydrogen sulphide and sulphurous gases must be removed, if they are suspected to exist, before the estimation is made of the CO, otherwise their presence would seriously interfere with the accuracy of the estimation.

Modifications of the mode of estimating the amount of CO from the above reactions have been founded either by estimating the amount of CO_2 or the amount of iodine liberated.

The method proposed by Gautier[4] is to absorb the CO_2 formed and to measure its volume after liberation with an acid.

The method of Nicloux[5] was to dissolve the liberated iodine in chloroform and estimate its amount colorimetrically.

Fillunger[6] described an apparatus devised by Karl Molterski and

[1] *Comptes Rendus*, 1898, Vol. CXXVI., p. 938

[2] *Arch. Hygiene*, Vol. XLVI., p. 284 ; *Jour. Soc. Chem. Ind.*, 1903, Vol. XXII., p. 652.

[3] *Chem. Zeitschr.*, 1888, Vol. XII., p. 1726.

[4] *Comptes Rendus*, 1898, Vol., CXXVI., p. 931 ; *Trans. Inst. Min. Eng.*, Vol. XVI., p. 571.

[5] *Comptes Rendus*, Vol., CXXVI., p. 1299.

[6] *Osterreichische zeitschr. f. Berg. und Huttenwesen*, 1903, Vol. LI., p. 216 ; *Trans. Inst. Min. Engin.*, Vol. XXXI., p. 713.

R. Nowicki in which, after the free iodine had been removed by metallic silver, the CO_2 produced was estimated by Pettenkofer's method. The instrument or apparatus used consists of the usual measuring glass, two absorption pipettes containing barium solution, drying tubes, a combustion vessel for the iodine pentoxide and silver, and a Winkler apparatus to burn the methane with cupric oxide. It is claimed for this method that it is more easily and rapidly manipulated than the Winkler apparatus.

Kinnicutt and Sanford recommend that the liberated iodine should be absorbed in a solution of potassium iodide and then titrated with $\frac{N}{1000}$ sodium thiosulphate, freshly prepared from a deci-normal solution.[1]

Probably one of the best methods of later years is that advocated by Chance as the result of experience.[2]

We give in substantial detail the steps of the method :—The sample of air is taken in a cylindrical copper vessel which has a tap at the top and bottom. Placed in a vertical position, the bottom tap is connected with a water-supply, so that when the tap is opened and the water rises in the vessel, the contained air is displaced through the upper tap which has been connected with a U tube, the first leg of which is filled with calcium chloride and the other with soda lime. From this tube the air passes into a second small U tube which contains 25 grammes or thereby of iodine pentoxide mixed either with glass wool or asbestos, to render it porous. This second U tube is surrounded by a sand-bath, the temperature of which is fixed between 150°C. and 160°C. From this the air passes to a Bowen absorption tube containing about 5 c.c. of a 10 per cent. solution of potassium iodide. When one or two litres of the suspected air have been passed at the rate of about one litre per hour, the lower tap of the sample vessel is closed, and when bubbles cease to pass through the absorption tube, the upper tap is next closed. To the absorption tube is now attached an aspirator, and from the exterior atmosphere a current of air is slowly drawn through the apparatus for about half an hour. The absorption tube is now disconnected, its contents rinsed out with distilled water and, after the addition of a few drops of starch solution, is then titrated with $\frac{N}{1000}$ solution of sodium thiosulphate, 1 c.c. of which is equivalent to 0·056 c.c. of carbon monoxide gas at normal temperature and pressure.

The quantity of air sample employed in the experiment being accurately known, and being reduced to normal temperature and pressure, the percentage volume of CO gas may now be calculated.

[1] *Jour. Amer. Chem. Soc.*, Vol. XXII., p. 14.

[2] *Jour. Franklin Inst.* Vol. CLXXII., No. 5, November 1911.

CHAPTER XVI.

APPARATUS FOR RESCUE WORK IN MINES AFTER UNDERGROUND FIRES AND EXPLOSIONS.

Any apparatus which is likely to prove of useful service in rescue work in mines during underground fires and after explosions of fire-damp, or, indeed, in any atmosphere which is incapable of supporting respiration, must be of such construction as will make the wearer independent of the atmosphere into which he has to enter, irrespective of its composition or character.

There are not a few contingent conditions in which the use of such an apparatus may prove of the greatest value. As has been said in previous chapters, accidents as those above indicated produce conditions of the atmospheres of mines which render them incapable of supporting the lives of men and animals, the chief of which, perhaps, may be divided into (*a*) those in which there is insufficient oxygen, as in black-damp, (*b*) those in which a poisonous gas, as carbon monoxide, exists in toxic amounts, as in after-damp, (*c*) those in which the air contains pungent vapours or gases, which render respiration difficult or impossible, as in smoke from fires, and (*d*) those in which there is a combination of toxic gases and pungent vapours.

In order to enable the wearer of any such apparatus to be independent of the air in such conditions, the apparatus must conform to certain well-defined conditions of construction, and be capable of producing a sufficient and self-contained atmosphere. Among the principal of these conditions are the following, viz. :—(1) the apparatus must, by its adaptation to the mouth and nose of the wearer, completely exclude the possibility of entrance of the irrespirable or poisonous air into which the wearer is to proceed ; (2) it must be provided with or be capable of generating within itself a sufficient supply of readily-available oxygen calculated to last for a given period of time ; (3) it must be provided with means whereby to remove from the expired air of the wearer the carbon dioxide gas and the bulk of the watery vapour, as these are exhaled while the apparatus is being used ; (4) a breathing-bag or reservoir wherein a supply of air may always be present during the use of the apparatus, which shall act as a buffer between the inspiratory and expiratory movements of the wearer ; (5) where the supply of oxygen is obtained from the compressed gas in cylinders, it must carry, in a position visible to the wearer, an indicator

which, when observed, will warn the wearer of the gradual use of the oxygen, so that he may be able to bear himself to safety; and (6) the apparatus ought to be portable, compact, and of reasonable weight, and when placed in position on the body of the wearer, the weight should be so apportioned to the body that it is distributed as evenly as possible, so as to prevent early or unusual fatigue.

Regarding these desiderata the following points may be noted. It will be obvious, unless the apparatus be air-tight at the points of contact with the breathing passages of the wearer, that irrespirable vapours or toxic gases in the atmosphere entered will readily find entrance into the lungs of the wearer and thus nullify his security from danger. Although such risks of immediate danger doubtless vary in some degree in different atmospheres, depending chiefly on the character of the toxic gas or vapour present in the local atmosphere, it is wholly unnecessary as it is also dangerous that any apparatus should be so ill-fitting as to expose the wearer to any such risks; besides, the possible entrance inwards of a gas or vapour involves also the likely leakage outwards of the oxygen upon which his period of stay in the noxious atmosphere depends. Particularly is such leakage serious when the wearer has to enter an atmosphere charged with pungent and irritating smoke or vapours. Hence any such apparatus must be so fitted to the head or breathing parts as to be air-tight.

Owing to the knowledge derived from experimental physiology, it is not now difficult to calculate the amount of oxygen required to be carried in such apparatus to satisfy the needs of a wearer for a given length of time, independent of any other supply of air, despite the toxicity or irrespirability of the air surrounding him. While an average man at rest consumes about 0·3 litre of oxygen per minute, that amount is increased correspondingly as he does any work and as is the amount of work done, up to about 2 litres per minute. Between the point of rest, therefore, and the point of severe exertion or strenuous work, the amount of oxygen consumed will vary between these two figures. Without a sufficient supply of oxygen, therefore, the wearer of any such apparatus would be incapable of maintaining his existence, much less of being able to render aid in rescue work.

In all the apparatus now on the market, the available amount of oxygen, however supplied, is such that for all purposes it is sufficient to last for a given maximum length of time. From a consideration of the details which follow respecting the supply of gas in each form of apparatus, it will be seen that more than enough oxygen for the time the apparatus may safely be worn is supplied.

It is not enough, however, merely to provide sufficient or even abundant oxygen: it is essential, indeed, that there should be on the

apparatus within easy vision of the wearer some indicator which will afford, when consulted, definite warning of the progress of consumption of that gas. In our view, it is imperative that such should be found on every form of apparatus, for without an indicator, and in the excitement of the hazardous work sometimes to be done, no reliance can be expected to be placed on the personal feelings alone of the wearer as a test with respect to his present and available supply of oxygen.

Since, moreover, such apparatus must be self-contained, it is necessary that it should contain provision whereby the carbon dioxide gas and the watery vapour of the expired air of the wearer is removed as it is exhaled, otherwise the air at his call during the currency of its use must become increasingly contaminated and progressively warmer, until eventually it would become intolerable, if not, indeed, in some measure dangerous to respire. It has been proved experimentally that air containing 5 per cent. of carbon dioxide causes distinctly hurried breathing, and that when increased to about 7 per cent., the breathing becomes distressing; but it has to be borne in mind that amounts even less than 5 per cent. in the warm motionless air of mines, when ventilation has broken down, will not only cause the respiration to be more rapid than normal, but will disable the person from doing ordinary and, still more, extraordinary work, for any length of time without serious bodily discomfort, almost amounting to pain and distress.

Respecting the out-put of carbon dioxide and watery vapour from the lungs of the average adult, it will probably be enough to say that their amounts steadily increase from the point of rest through easy work until severe labour is being performed, and that during this last stage, if continued during two hours, the total amount of the gas exhaled would be about three and a half cubic feet. Provision must be made, therefore, in the apparatus supplied with oxygen gas for the absorption of both of these products of respiration.

This is accomplished in several forms of apparatus by causing the expired air to pass over or through some chemical absorbent in its circulatory course through the apparatus, whereby the former CO_2 especially and the latter watery vapour to some extent are removed. Experimental work with different forms of apparatus now in use has proved that the absorbents employed are fairly effective for their purpose. In some of the apparatus, besides, contrivances have been devised to diminish the heat contained in the expired air, thus rendering the air after admixture with fresh oxygen cooler for use over again.

In order to maintain continuity and to diminish resistance in the respiratory movement within the apparatus, it is essential, also, that a breathing bag or air-reservoir should form part of it, so as to act as a reservoir for the tidal movement of the air within the apparatus. This commonly

consists of one or two moderately-distensible rubber bags, the capacity of which must be sufficient to contain a little more than the largest volume of air which may be given off in the deepest expiration or may be taken in during the deepest inspiration.

In connection with such apparatus there is another point which calls for attention. When a person, without any apparatus affixed to his air-passages, breathes in the open in a pure atmosphere, for the most part he empties his lungs and air-passages of the impure air at each expiration, and taking a fresh inspiration, he draws into his lungs a new supply of fresh air. But the conditions are somewhat altered in the case of a person over whose air-passages an air-tight apparatus has been applied, in respect that when he finishes the act of expiration into the apparatus, his principal air-passages remain more or less full of the expired air, rich in CO_2 and watery vapour, and this portion of air becomes the first portion of the next inspiration of air which he takes from the apparatus. This space between the lips and the lungs has been called the "dead-space." In the case of the wearer of an apparatus of this kind, therefore, the size or volume of this "dead space" becomes of importance, since it varies in size and volume in different apparatus on the market, owing to their different constructions. The larger this "dead-space" is, the larger obviously will be the volume of impure air taken into the lungs of the wearer at each new individual inspiration. Experiment has shown that in any form of apparatus this space should preferably not exceed from 200 c.c. to 250 c.c. at most.

Since any such apparatus must be borne by the wearer, in order that it may prove of the greatest utility it ought to combine reasonableness in weight and compactness in form. The burden of carrying a heavy apparatus while overcoming obstacles met with underground, such as crawling through confined openings or surmounting falls, etc., and not infrequently amid immoderately-heated atmospheres, is apt quickly to become a tiresome load, and, therefore, to limit the useful work which a wearer might perform. For like reasons, the apparatus must be as compact as possible, must fit well to the body, and must present the fewest projecting or angular parts, so that it may not be dislocated from its position or be unnecessarily exposed to injury, while the wearer is attempting, perhaps, to crawl and squeeze through a comparatively narrow space.

Much ingenuity has been expended by inventors in trying to achieve these desiderata, and it is not improbable, where cylinders of gas have to be carried, that almost the lowest limits of weight have already been attained. When it is considered that these cylinders are the source of supply of the required oxygen, and that they must be constructed to withstand a very high internal pressure, it is, perhaps, too much to hope

that any substantial reduction of weight will be obtained; indeed, it would appear as if the total weight had reached an irreducible minimum.

Sir W. E. Garforth, the deviser of one of the forms of apparatus to be described, has set down in detail[1] what, in his opinion, are the desiderata in an efficient breathing-apparatus, as follows:—"The rescue apparatus, to be of real practical value, should not be too heavy or cumbersome, and should have its weight distributed over the various parts of the body; an automatic arrangement for the supply of oxygen is essential, as it is often absolutely vital that an explorer should have the free use of both hands; two oxygen cylinders with separate valves are necessary, one for use in advancing and the other for retreating; the gauge recording the pressure of oxygen in the cylinders should be so placed that the wearer can read it either direct or by means of a mirror placed within the protecting cover of the gauge, so that in case he should find himself alone he would immediately be able to determine what amount of oxygen he may have left to enable him to reach a place of safety. The apparatus should allow the head full freedom, and the wearer should be able to breathe in a natural way, not with the nose clipped or the mouth gagged. The mouth piece should be so constructed that it may easily be slipped on and off, so that whenever fresh air is encountered, advantage may be taken of it and a corresponding quantity of oxygen saved. The eyes should be protected by goggles for use in smoke, but these should be so arranged that they can be easily removed independently of the helmet, as they are often unnecessary in after-damp. Each apparatus should be capable of supplying the wearer with air as required, for a period of at least two hours whilst engaged in laborious work, or for four or five hours if the physical work is easy, as when taking readings of a thermometer in a heated and noxious atmosphere; the cylinders containing the oxygen should be sufficiently strong to withstand a working pressure of 120 atmospheres (1,800 lbs. per square inch), if the generator type of apparatus be used. The chemicals used for absorption of the carbonic acid gas should be of the very best quality, and not deteriorated by previous exposure to air, and a large surface should be exposed in the purifier for active chemical combustion."

In the description of the different forms of apparatus which follows, we have given the total weight of each form.

Classification of Kinds of Apparatus Available.

The apparatus now on the market for rescue and aid work may be divided into three groups, as follows:—

[1] Garforth: *Suggested Rules for Recovering Coal Mines after Explosions and Fires*, p. 26.

1. Those in which the oxygen is provided as compressed gas in cylinders.
2. Those in which the oxygen is generated within the apparatus from contained chemical substances.
3. Those in which the oxygen is provided in the form of liquid air.

In the first group will be found the largest number of apparatus. It includes the following, viz. :—(*a*) Esseff, (*b*) Draeger, (*c*) Proto, or Fleuss or Fleuss-Davis, (*d*) Shamrock, (*e*) WEG, and (*f*) others, as the Meco. In the second group there is, at present, one or two apparatus, mainly in this country, viz. :—the "Pneumatogen" apparatus, and the Hall-Rees.

In the third group there is, so far as we know, one only, viz. :—the "Aerolith" apparatus.

Within the apparatus belonging to the first group there are individual differences within narrow limits in the mode of supplying the oxygen, but these will be pointed out in the descriptions of the individual apparatus which follow.

First Group :—The following is a brief description of the different apparatus named in this group.

I.—Esseff.

The name of this apparatus is derived from the pronunciative value of the initial letters of the name of the company—Sauerstoff-Fabrik Company of Berlin—who are the makers of it.

It consists of the following working parts, viz. :—(*a*) oxygen cylinders, each of which contains between 125 and 150 litres of pure compressed gas ; (*b*) a pressure-reducing valve, by which the flow of oxygen from the cylinders into the breathing-pipe is controlled and regulated ; (*c*) an injector or circulating nozzle which, connected with the reducing valve, allows the oxygen to flow at the rate of two or three litres per minute, and determines the flow of expired air from the wearer into the absorbing chamber : (*d*) an absorbing chamber, which contains caustic potash cartridges and some infusorial earth for the purpose of taking up the CO_2 and watery vapour from the expired air ; (*e*) breathing-bags, for regulating the movement of air during inhalation and exhalation ; (*f*) a pneumatic pump, which can be operated by the wearer when needed ; (*g*) connecting tubes or pipes, which join together the different parts of the apparatus in which the respiratory cycle takes place ; and (*h*) a mouth-piece or helmet, as the case may be, depending on the mode of affixing the apparatus to the wearer.

The following figures illustrate the different parts and disposition of this apparatus.

Fig. XVI. is a diagrammatic representation which shows the arrangement of the different parts to one another.

Fig. XVII. shows the apparatus *in situ* on the back of a wearer, without the mouth-piece being adjusted; Fig. XVIII. shows the entire apparatus in position, the mouth-piece being in its place; and Fig. XIX. indicates the apparatus in position with helmet attachment, and shows the distribution of the apparatus on the body.

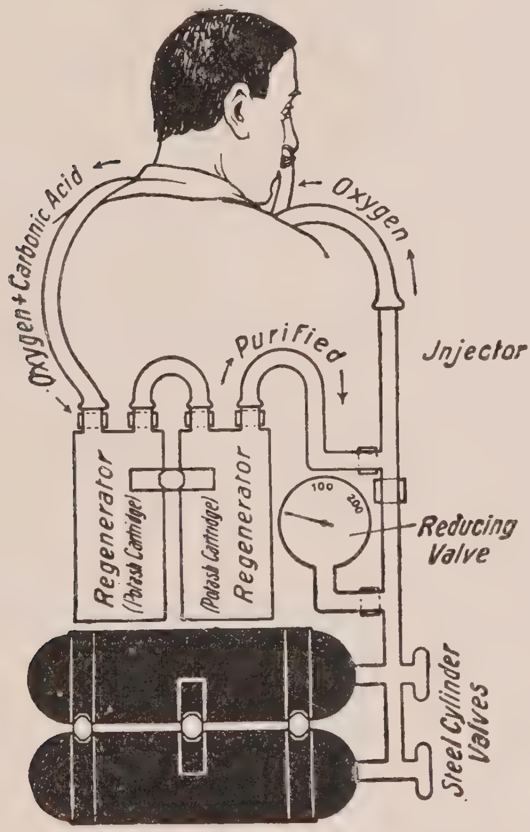


Fig. XVI.

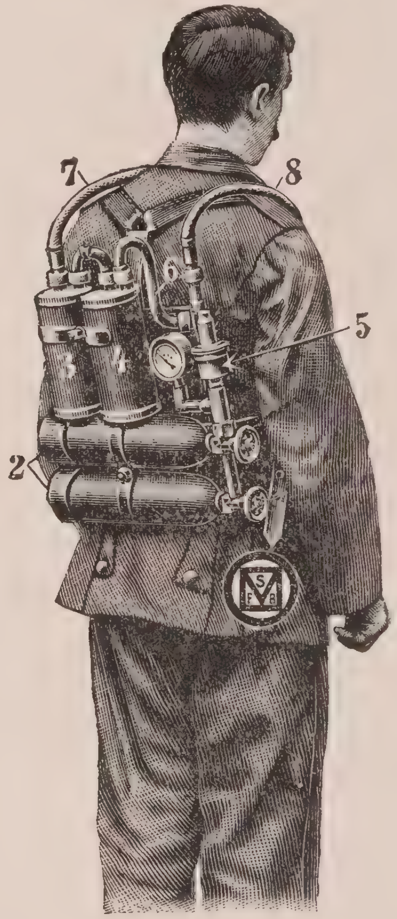


Fig. XVII.

Fig. XVI.—The respective parts of the apparatus are named in the Fig. and the cycle of the respiratory movement within it is indicated by the arrows. The rounded black cylinders contain the oxygen.

Fig. XVII.—This shows the back view of the apparatus as placed on the wearer. The figure 2 indicates the oxygen cylinders; 3 and 4, the potash cartridges; 5, the circulating nozzle or injector; 6, the connecting tube between the potash cartridges and the oxygen pipe, by which air purified of CO_2 and moisture passes into the latter from the former; 7, the tube conveying the exhaled air from the helmet of the wearer to the potash cartridges; and 8, the pipe for supplying oxygen and purified air to the helmet of the wearer.

The cycle of movement of air within the apparatus may easily be followed from the description of Fig XVI. From the mouth of the wearer the expired air passes to the breathing-bags, thence by the tube, marked 7 on Fig. XVII., which in Figs. XVIII. and XIX. is shown passing over the left shoulder to the potash chambers, designated

"regenerator" on Fig. XVI., where it is purified of its CO_2 gas and watery vapour, thence purified it enters the tube, marked 8 on Fig. XIX., into which is passing the oxygen from the cylinders, and mixing with

Fig. XVIII.

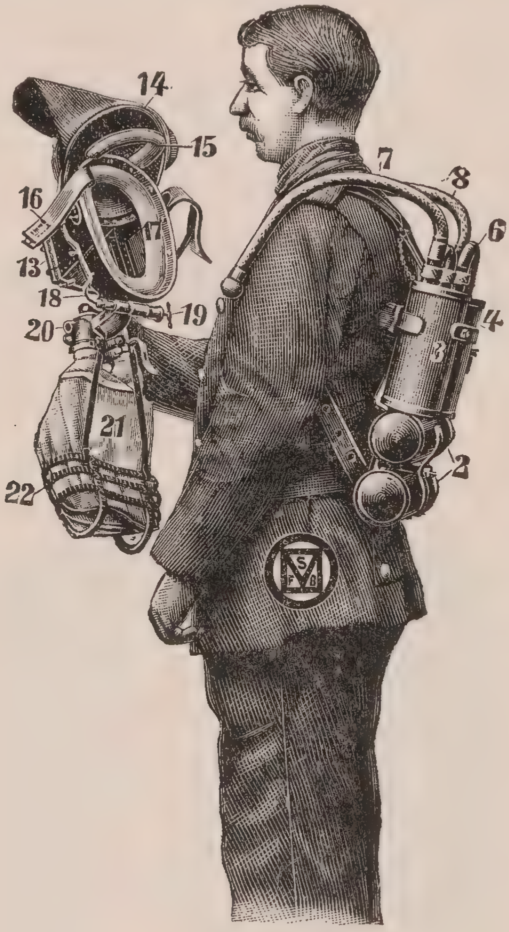


Fig. XIX.

Fig. XVIII.—The figures 2, 4, 5, 6, 7, and 8, bear the same references as on Fig. XIX. ; 9, indicates the valve box containing inhaling and exhaling valves, and shows connecting tubes to inhaling and exhaling valves ; 9*a*, the mouth-piece ; 10, the breathing-bag ; 13, the goggles or eye-protecting glasses ; 14, the nose-cap for producing a gas-tight closure of nose from outer atmosphere.

Fig. XIX.—In this Fig. the only explanatory figures necessary, as the others have been explained in the previous Figs., are 13, the open window of helmet ; 14, the head leather strap, which is secured by means of the neck-strap ; 15, the padded head support to make the helmet fit surely and comfortably ; 16, the head-strap by which the helmet is buckled at the back of the head ; 17, the pneumatic rim of helmet adaptable by inflation to any shape of face so as to be gas-tight ; 18, tube for conducting the air from the air-pump to the pneumatic rim ; 19, pneumatic pump, by which pneumatic rim may be adjusted by wearer ; 20, the valve box, made of light metal and easily dismounted for repairs ; 21, the breathing-bags for inhaling and exhaling, made of good, durable rubber ; 22, the basket-work guard for the breathing-bags to prevent compression and damage to bags while wearer is working.

the oxygen, is moved onwards by the injector to the breathing-bags, and thence to the air-passages of the wearer.

The weight of the apparatus varies with the extent of the equipment, the variations being due to whether one or two cylinders are used in the

pattern. With the mouth-piece pattern the weight runs between 25 lbs. and 34 lbs., and with the helmet pattern between 27 lbs. and a little over 36 lbs., with one or two cylinders respectively.

II.—Draeger.

Description of Apparatus.—As in the former apparatus, this is borne on the shoulders, the weight being distributed over the front and back of the chest. The back and front parts are connected by two

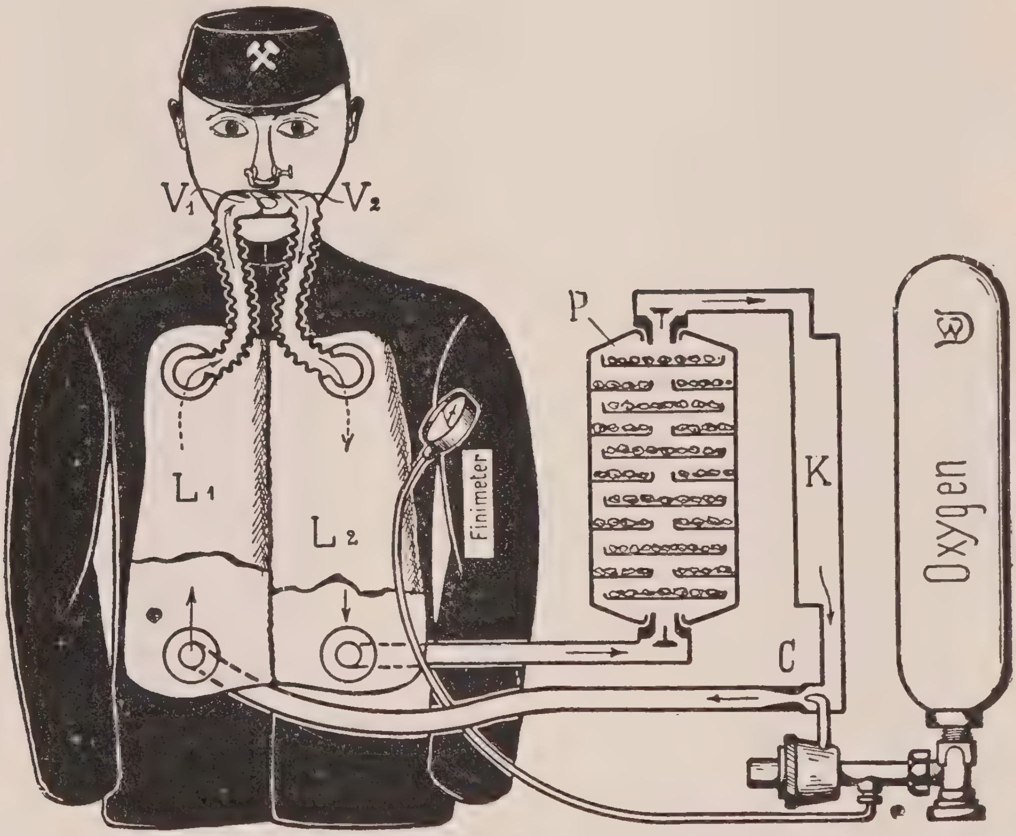


FIG. XX.

Description of Figure XX.—*P* represents the alkali cartridge contained in the cartridge carrier; *K*, the cooler; *C*, the aspirating pipe or injector; L_1, Breathing-bag for purified air; L_2, Breathiug-bag for expired air; V_1, Valve admitting purified air to wearer; V_2, Valve permitting exit of expired air from mouth of wearer; the nostrils are closed by means of an air-clip.

flexible circulation pipes. The back portion consists of the following parts:—(*a*) the supporting frame with the back rests, the protecting arched rods, the cartridge carrier, the automat, and the cooler, (*b*) the potash cylinder, and (*c*) the oxygen cylinder. The two latter are detachable. The front portion consists of (*a*) the smoke helmet, (*b*) the mouth-breathing attachments, and (*c*) the two breathing-bags. It will be observed, therefore, that the back portion has to do with the purification and re-oxygenation of the air, and the front portion with the breathing of the wearer. Some details may now be offered regarding these constituent parts.

1. Back Part of Apparatus.

1. *Supporting frame.*—Nothing explanatory need be said respecting the action of the back rests or of the protecting arched rods, as these explain themselves, but attention must be drawn especially to the *automat* or that portion of the mechanism by which the respiratory process is regulated while the apparatus is being worn. Perhaps, indeed, of the mechanical parts of the machine it is the most important. It comprises (*a*) the finimeter, (*b*) the pressure-reducing valve, (*c*) the safety valve, and (*d*) the injector.

The *finimeter* is a high-pressure gauge, which indicates the quantity of oxygen contained in the steel cylinder. From the pressure indicated thereon, the quantity of gas in the cylinders may be read during the working period. The *reducing-valve* lowers the pressure of the oxygen in the cylinder to a constant and uniform working-pressure for the injector. The *safety-valve* acts to liberate over-pressure in the event of the working-pressure being at any time exceeded, and is provided for use only under those circumstances. The *injector* operates as the motive power for keeping the air in circulation within the apparatus while working, the motive power being produced by the oxygen flowing from the cylinder through the contracted nozzle, which forms the injector, thus converting pressure into movement. By its action it mixes the expired air from the wearer, purified of its CO_2 gas, with fresh oxygen from the cylinder.

The amount of air set in motion by the injector should amount to one and three quarters cubic feet per minute at least, and the measured amount of oxygen supplied by the injector, which is measured automatically, should be not less than 120 cubic inches per minute. In the standard pattern of apparatus, it is impossible for the delivery of oxygen to be interfered with either by accident or design. The valve of the oxygen cylinder enables the apparatus to be set in full action, or to be entirely stopped, or to act at any point intermediate to these. The *cooler* serves the purpose for which it is named. During the operation of breathing within the apparatus the liberated CO_2 gas and moisture in the expired air, being absorbed by the substance in the cartridge carrier, give off heat, hence the effluent air is warmed. During further continuance of use, this warmth increases until the air may become uncomfortably warm, but owing to the action of the cooler, the temperature of the moving air is reduced and maintained at a comfortable warmth. For the purpose, moreover, of still further reducing the temperature of this effluent air, two breathing-bags were introduced in the 1913 pattern of apparatus.

The absorbing material employed in this apparatus to take up the

CO_2 gas and moisture is placed in the potash cartridge, and fills about twenty shallow trays. By arrangement of baffles in these trays, the exhaled air is made to traverse the whole of this material, and thus absorption is facilitated. Each cartridge has a detachable label on which the initial weight of the charged cartridge is printed, so that any increase in

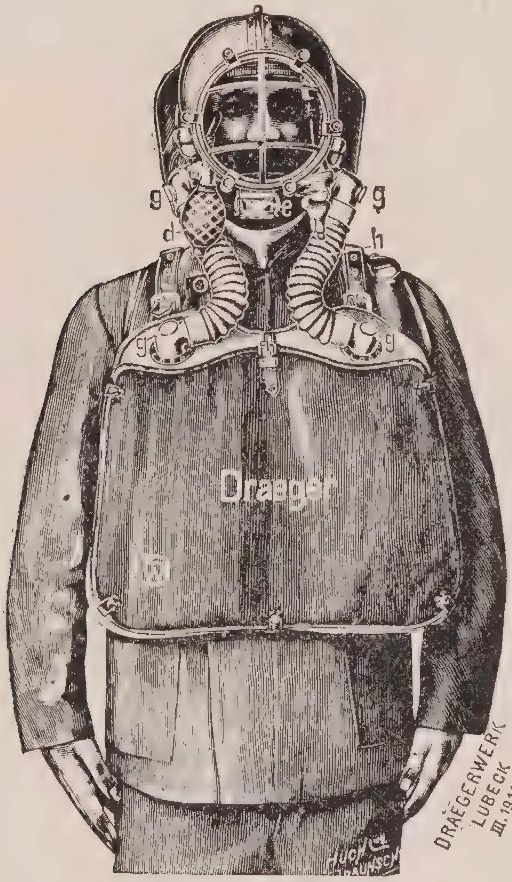


Fig. XXI.

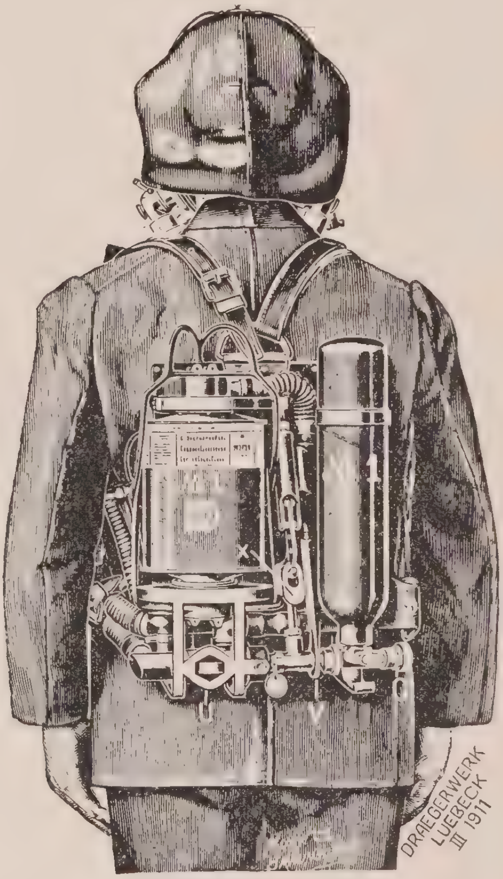


Fig. XXII.

Fig. XXI.—Draeger two-hour apparatus. Front view of apparatus adjusted to body of wearer; with helmet.

Description.—d = moveable corrugated flexible breathing-tubes; g = pipe or tube-couplings. The pocket at f contains the finimeter. The breathing-bag covers the entire front of the chest.

Fig. XXII.—Draeger apparatus. Back view, showing oxygen cylinder and cartridge-case. c = the injector; v = spanner for oxygen cylinder; w = hexagonal socket for unscrewing locking-nuts of reserve cylinders.

weight from use can readily be ascertained. When a cartridge is fresh, it gives off a rattling sound when shaken, but after use, owing to the absorption of the moisture from the lungs, this rattling becomes less and less distinguishable.

The substance contained in the cartridges is a mixture of caustic

potash and caustic soda in the form of small granules, the proportions being one-third of the former and two-thirds of the latter. It has a porous, crystalline structure, and thus enables the process of absorption to be more readily achieved, since both the CO_2 and moisture are to be absorbed.

The oxygen cylinder contains oxygen compressed at 150 atmospheres.

2. Front Part of Apparatus.

The part of the apparatus which is carried on the front of the chest of the wearer is composed of the following parts:—(*a*) smoke helmet, (*b*) mouth-breathing attachment, and (*c*) breathing-bags.

1. *Smoke-helmet.*—This is made up of the following parts, viz.:—(*a*) pneumatic tube with bulb inflator, (*b*) an air escape-valve, (*c*) a leather neck-guard and straps, (*d*) a window and lattice, (*e*) an air-valve, (*f*) a window-cleaner, and (*g*) two mica-plate valves. Of these, attention need only be specially directed to the mica plate respiration valves. These are connected with the breathing-bags, one with the bag into which enters the expired air, the other with the bag from which proceeds the purified air. The resistance of these valves to the passage of air during breathing is only from one-twelfth to one-eighth of an inch of water by gauge.

2. *The Mouth-breathing attachment.*—This may be used instead of the smoke-helmet. The actual mouth-piece is made of rubber. When in use the nostrils are closed with a nose-clip, and the eyes are protected by smoke-goggles.

3. *Breathing-bags.*—These are two in number. One is for the purpose of receiving the exhaled air from the lungs of the wearer, and the other for holding the purified air and for passing that on to the wearer. They communicate with each other by means of a relatively narrow passage, so that whenever an unusually large supply of air is needed by the wearer, the contents of both bags may be called upon to supply the extra amount of air. A *blow-off valve* is also provided in connection with the bags to enable any surplus air to escape, if and when the bags become over-distended.

The reader will, perhaps, follow more readily the foregoing description by referring to Fig. XX., which is a diagrammatic drawing of the apparatus applied to the person. If it be assumed that the apparatus has been adjusted, it is clear that the respiratory organs and passages of the wearer are now completely shut off from the external atmosphere, and have become an extended part of the apparatus itself. Under these circumstances, since air from the outside is no longer admitted, there are only two forces within the apparatus by which the movement of air may be effected and maintained, viz.:—(1) the action of the injector

C, and (2) the respiratory action of the wearer. The injector operates by the power generated from the escape of the compressed oxygen from the cylinder and the narrowed orifice of the tube in front of the oxygen cylinder tube.

Between the lungs on the one hand and the injector on the other, therefore, are the two breathing-bags, L_1 and L_2, or air-reservoirs, which also act as a compensating buffer between the rhythmic respiration and the continuously-acting circulation within the apparatus induced by the injector. If it be supposed that the apparatus has been adjusted to the face of the wearer, and that the injector has already been regulated to pass oxygen from the cylinder at the rate of 120 cubic inches per minute, the course of the air-circulation within the apparatus will now be as follows:—The exhaled air passes from the mouth by the valve V_2 in the direction of the arrow into the breathing-bag, L_2, and by the lower valve of the breathing-bag, L_2, into the upper tube, proceeds along this tube in the direction of the arrows, then enters the cartridge carrier, *P*, where it encounters the alkaline absorbent, by means of which its contained CO_2 and moisture are removed during its upward passage through and between the trays. It continues to pass forward in the direction of the arrow through the cooler, *K*, where it loses some of its heat, then moves onward to the bottom of the tube, and at the point, *C*, it receives further impetus onwards as well as a further supply of fresh oxygen, by the action of the injector, finds itself temporarily at comparative rest in the breathing-bag, L_1, and sooner or later is passed therefrom by valve V_1 into the mouth and lungs of the wearer as required; and so on *da capo*.

The finimeter or end-measurer of the oxygen-supply is placed in sight of the wearer.

It is to be remembered that the alkaline cartridge and the oxygen cylinders are calculated to last for a certain period of time only, and that whereas the latter may be replaced or refilled with fresh oxygen, the spent cartridge material and the cartridge itself have to be removed, and freshly charged cartridges substituted With the pattern of 1913, however, both the oxygen cylinder and the potash cartridge may be replaced, even in a smoke-laden atmosphere.

As set up, this apparatus is able to purify about 3000 litres or 105 cubic feet of air, to supply 120 litres or four and a half cubic feet of oxygen, and to absorb 50 litres or one and a half cubic feet of CO_2, per hour. One potash cartridge, No. 2 of the 1912 pattern, enables an amount of work equivalent to 260,000 foot-pounds to be performed.

The complete apparatus, fitted with smoke helmet for use in mines, weighs 41 lbs, and with the mouth-breathing fitting instead of the helmet, 37½ lbs.

III.—The Proto, or Fleuss, or Fleuss-Davis Apparatus.

The Fleuss apparatus was the forerunner of all the forms of rescue apparatus in which compressed oxygen was employed. In its more modern form the supply of that gas is continued from such a source. The cylinders in the "Proto" apparatus have a delivering capacity of 2 litres per minute, and, besides, the supply of oxygen may be regulated by hand according to the fulness of the breathing-bag.

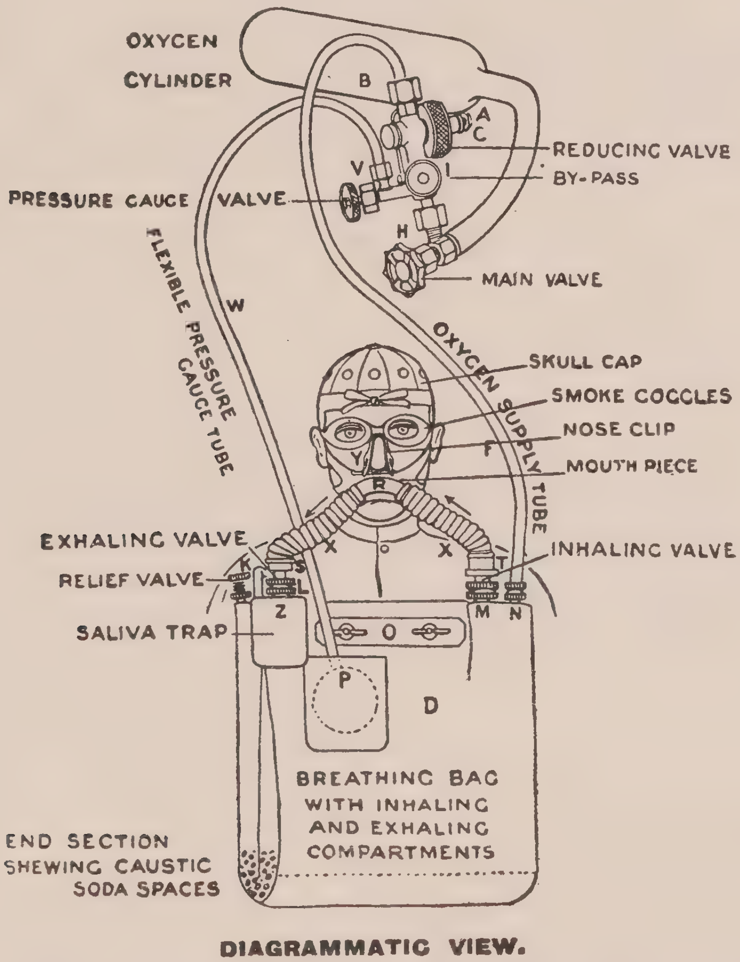


FIG. XXIII.—DIAGRAMMATIC VIEW OF THE "PROTO" APPARATUS.

The absorbent in this apparatus is caustic soda in the form of sticks, the alleged advantage of this form being that the sticks afford a larger absorbing surface, remain longer dry, and may be broken when required during use of the apparatus to expose fresh absorbing surfaces, by forcibly shaking or agitating them in their case.

In the newer form of apparatus, moreover, the breathing-bag has been enlarged, the adjustable blow-off valve is inserted in the breathing-bag, thus enabling any excess of expired air to escape should, perchance, the bag become hyperdistended, the mouth-piece has been improved and

has been fitted with a saliva-catcher, the inspiratory and expiratory valves have been placed in a position close to the breathing-bags, the oxygen cylinders have been fitted with a reducing-valve which is set to deliver 2 litres per minute, and there is a bye-pass by which a larger supply of oxygen may be turned on when needed.

Besides, the pressure-gauge has now been brought round to the front, so that the wearer of the apparatus may be able to watch it.

The weight of the new pattern, fully charged with oxygen and absorbents, is a little over 30 lbs.

The form of the apparatus will, perhaps, be better understood by reference to Fig. XXIII. The figure may be described as follows :—

The oxygen cylinders, *B.B.*, are charged through the nipple, *H*, with the gas at a pressure of 120 atmospheres or about 1,800 lbs. per square inch. To the cylinders thus charged, the reducing valve with its tubes, etc., are attached to the nipple at *H*. These cylinders contain jointly about 10 cubic feet of oxygen at above pressure, and this amount is sufficient for fully two hours' use. Should the cylinders be charged at less pressure than that named, their contents will last for correspondingly shorter periods. The breathing-bag, *D*, composed of two compartments, is charged by putting into it 4 lbs. of stick caustic soda, half of that amount being placed in each compartment of the bag. When charged, it is at once fastened to the rest of the apparatus by the clamps and wing nuts, *O*, but if the apparatus is not to be used immediately, then its contents are excluded from the air by plugging the mouth-piece of the bag with a solid rubber plug, which is supplied with the apparatus. When the apparatus is being fitted on a wearer for use, the inlet or inhaling valve, *T*, and the outlet or exhaling valve, *S*, must be tightly screwed so as to exclude air. The relief-valve, *K*, is only to be opened by the finger of the wearer when the breathing-bag becomes unduly inflated through excess of oxygen and air. This may happen from time to time, as the amount of oxygen delivered by the reducing valve is always rather more than the wearer actually requires for respiratory needs.

The rest of the parts explain themselves in the figure.

The whole apparatus is supported on the body of the wearer by a broad belt with three straps and buckles, and to enable the wearer to crawl with the apparatus fixed to his body, the bag is kept in position by a pair of leather loops on the body side of the bag. Further, the bag is slung on the body by means of a pair of shoulder-braces with adjustable straps and buckles at the back. When the wearer is about to don the apparatus, he places the equipment over his shoulders, fastens the belt, and takes the rubber plug out of the mouth-piece of the breathing-bag. At the moment the mouth-piece is placed in the mouth, or the mask is adjusted over the face, the main valve, *H*, is opened by not more than one turn of the screw, the bye-pass, *I*, also for a moment or two, in order to partially inflate the breathing-bag, *D*, for a start. When this is done, the bye-pass is screwed air-tight, not to be touched again while the apparatus is being worn, unless the breathing-bag should, perchance, become deflated owing to the valve, *C*, getting out of order. Breathing will now be comparatively easy.

THE "PROTO" OR FLEUSS APPARATUS.

Fig. XXIV.—Front view of the "Proto" apparatus. Mouth-piece and nose-clip type. Showing breathing-tubes and breathing-bag.

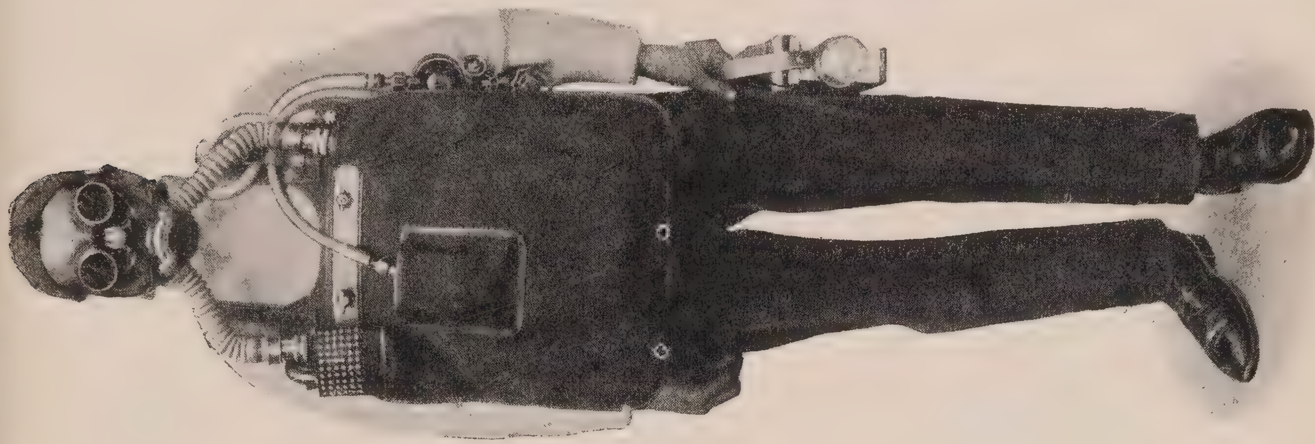


Fig. XXV.—Side view. Shows wearer reading the pressure gauge.

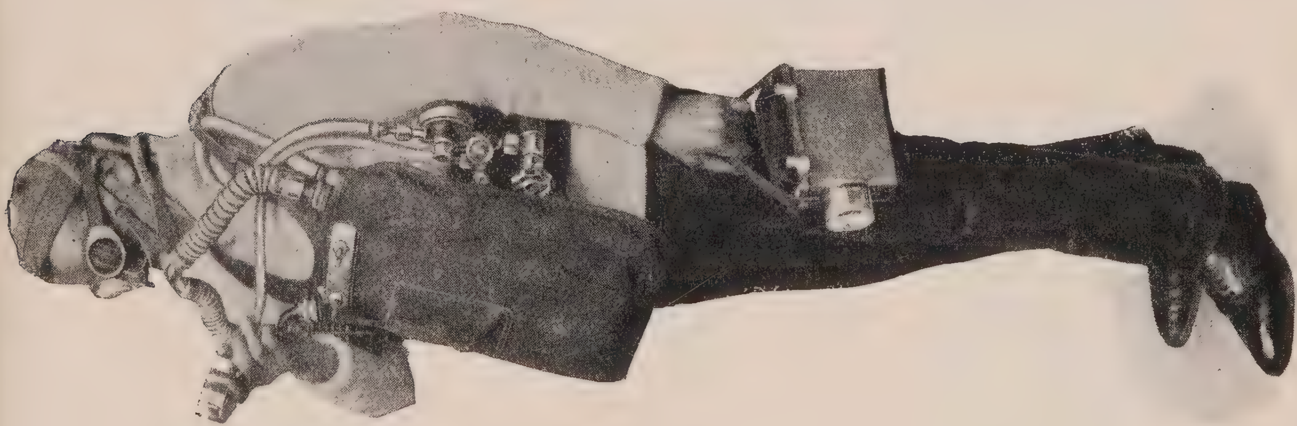
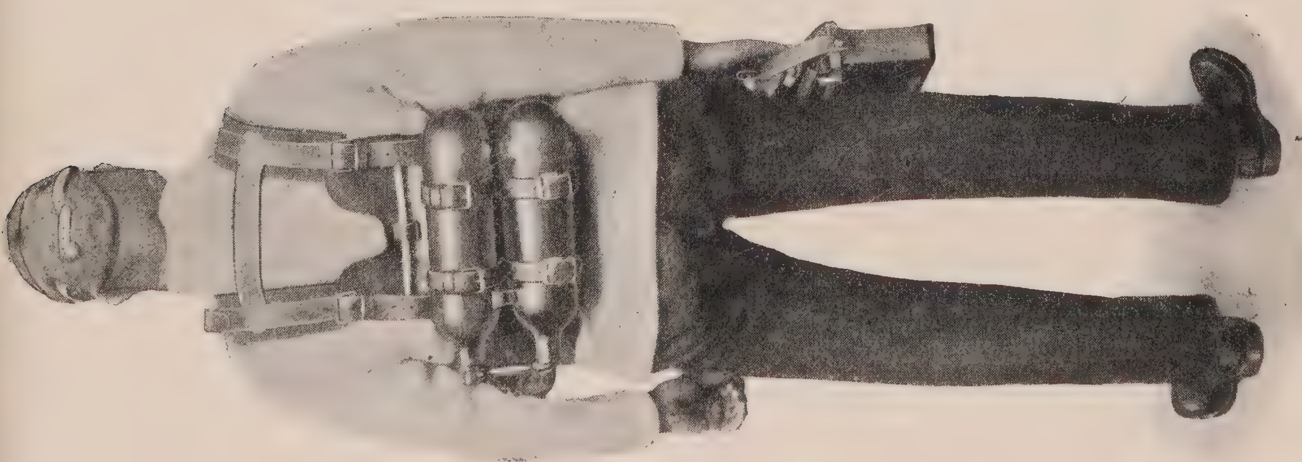


Fig. XXVI.—Back view. Showing position of oxygen cylinders.



It is advisable that each user of this apparatus should have his own mouth-piece and nose-clip or mask, firstly for easy fitting, and secondly for sanitary reasons.

When the use of the apparatus is over, the regenerator should be emptied of *all* its caustic soda contents, assisting the process by using warm water, the inside of the bag being thereafter dried with a clean cloth.

The "*Salvator*" apparatus, constructed under the same patents and on the same principles, is intended for any kind of suitable work not demanding more than one hour's time.

For use in smoky atmospheres, or atmospheres charged with irritating fumes, irrespirable under other circumstances, there is also provided by the same makers a smoke helmet fitted with an air-pump bellows and hose-piping connected with the helmet, whereby ordinary pure air may be supplied to the wearer of the helmet while he is at work.

Alliot has recorded the facts relative of the re-opening of the Norton Colliery after the great explosion in that pit, in which the apparatus just described was used without a single hitch.[1]

IV.—"Shamrock" Apparatus.

In this apparatus, like those already described, the motive power of the air circulating within it is the oxygen which is permitted to enter from the cylinder through the injector. With the expiratory tube open to the air, about eighteen litres of gas per minute are passed along the inspiratory tube, the suction action at the open end of the expiratory tube amounting to a pressure equal to a column of water eight centimetres in height. The apparatus, which weighs close upon 36 lbs., is distributed when in position between the front and back of the body of the wearer, the principal weight, 26½ lbs., being carried on the back. The two oxygen cylinders alone weigh 13 lbs. The absorbent used is potash, presumably in the form of caustic potash. It appears to contain about 76 per cent. of KHO, the remaining parts per cent. being for the most part of water. From experiments made with the air from the apparatus after use, it does not appear that the carbon dioxide is as well absorbed as it might be.

The apparatus consists of the following parts :—(*a*) oxygen cylinders ; (*b*) pressure-reducing valves with gauge and injector, (*c*) regenerator with 4-way-cock, and (*d*) smoke-helmet or mouth-piece with respiration-bag. The mechanical attachment of these cylinders to the rest of the apparatus has some differences singular to it, which, however, need not now be described.

[1] *Trans. Inst. Mining Engin.*, June 1913.

As the functions of these chief parts have already been discussed, it is unnecessary to recapitulate; but some mention may be made of the pressure gauge. This gauge bears a scale which enables the wearer of the apparatus to read off his loss of oxygen. On this scale there are red and black figures, the intention being that when one cylinder only is

"EVERTRUSTY" SHAMROCK APPARATUS.

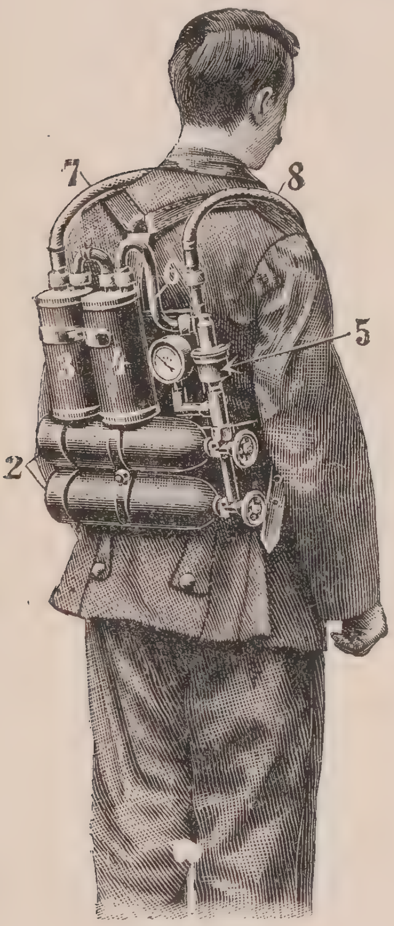


Fig. XXVII.

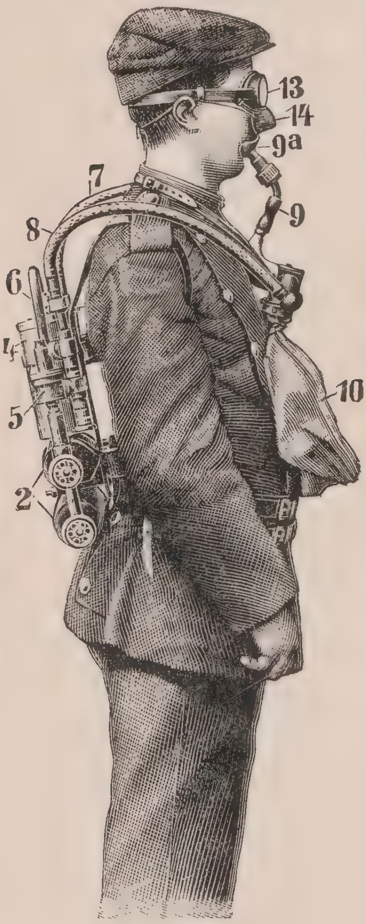


Fig. XXVIII.

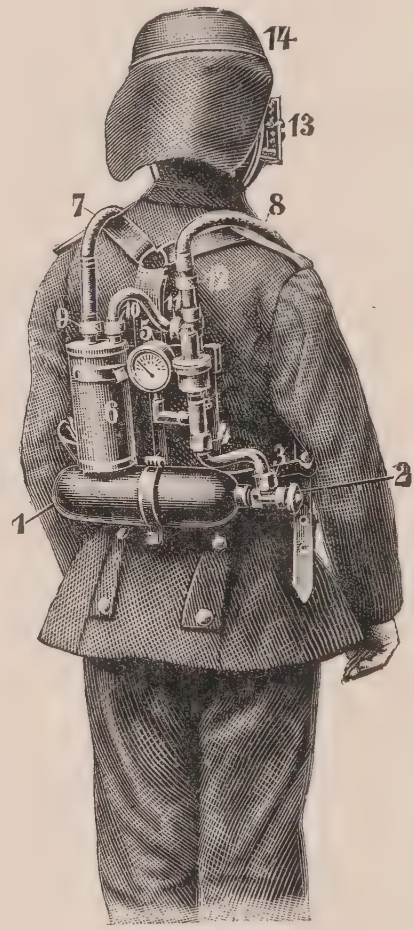


Fig. XXIX.

General description of Figs. XXVII., XXVIII., XXIX.—*2*=oxygen cylinders; *3* and *4*=potash cartridges; *5*=injector; *6*=tube containing purified air from potash chamber passing to inhalation tube; *8*=tube from mouth, carrying impure air to potash chamber; *7*=tube carrying purified air and oxygen to wearer; *9*=mouth-piece holder; *10*=breathing-bag; *14*=nose-cap; *13*=smoke-goggles. On Fig. XXIX., *14*=helmet.

Fig. XXIX.—Shows adjustment of apparatus with helmet.

supplying the oxygen, the red figures are to be used in reading, but when both are in use simultaneously, the black figures only.

In the latest pattern of machine, the absorbents employed in the regenerator are soda lime and caustic potash.

The smoke helmet is adjusted so that the upper covering of the helmet comes over the back part of the head, and when this is so placed, the helmet is then pulled to the front. The neck-leather has a strap

with a loop which is drawn tight. The window in front of the helmet should be kept open while the helmet is being adjusted, until just before the wearer is about to enter the mine or other place of irrespirable atmosphere. Inside the helmet is a pneumatic air-cushion which can be inflated so as to make the whole fitting air-tight. The inflation of this cushion is done by an inflator. The valve is on the helmet, and the helmet is united to the respiration-bag and the hose or tube connections. The pipe or hose which conveys the air to be inhaled is connected to the left side of the valve-piece, and that which carries the exhaled air to the regenerator or purifier is connected on the right side. The breathing-bags are fastened to the body by means of a bayonet belt.

Before the window of the helmet is closed, the oxygen cylinder and the regenerator must be opened.

Instead of the helmet, however, the mouth-piece fitting may be used. A movable rubber mouth-piece is fixed to the valve, and this is placed in the mouth. In order to prevent it from falling out of the mouth, it is surrounded with a wide rubber sheet. The air is then inhaled through the mouth-piece. The body of the valve is fitted with leather straps, by which it is held in position round the neck of the wearer. When the mouth-piece is used, the nostrils in this apparatus, as in the others similarly used, must be absolutely closed by means of a nose-clamp or cap. It is better also that the eyes should be protected by goggles.

The mouth-piece has the same connections with the hose or pipe as the helmet.

V.—The "WEG" Apparatus.

This apparatus has been designed and made by Sir W. E. Garforth, from the initials of whose name it derives its title. Like those already considered, the WEG apparatus is a compressed oxygen apparatus, but it differs from these in that the oxygen is drawn from the cylinders only when required, and in proportion to the immediate requirements of the wearer; that is to say, should the wearer be making violent physical exertion a larger amount, or should he be resting or using but little exertion, a smaller amount would be drawn.

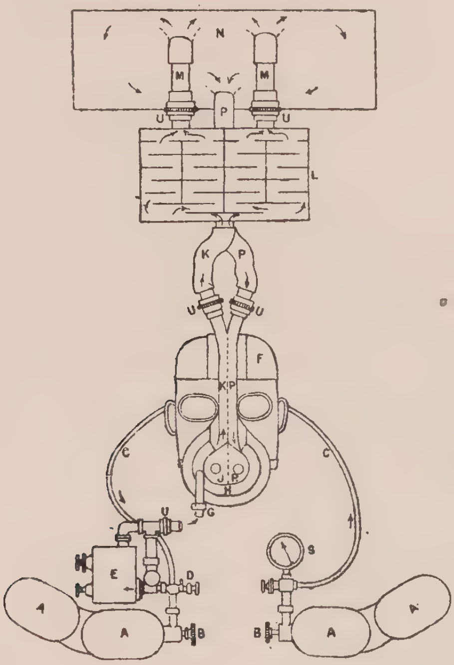
This is achieved by an ingenious arrangement or combination of two Beard's reducing-valves arranged in series, by which the negative pressure produced in the mask of the apparatus in the act of inspiration causes the oxygen to be delivered into the breathing-space. Moreover, there is an arrangement by which the end of the oxygen-pipe may be taken directly into the mouth of the wearer, and a full charge of oxygen thereby drawn in by the act of inspiration, and, in addition, a bye-pass for use should the valve by any chance refuse to act. The cylinders contain

jointly 160 litres or 5·3 cubic feet of gas, and they are so shaped that they conveniently fit to the body for carrying under the arms.

The absorbing material is caustic potash.

The entire apparatus is mounted on a strong canvas jacket which the person about to use it may readily adjust to his body. The weak part of the apparatus is said to be the head-piece or mask, in that it is not readily adjustable to different persons. This, however, is easily rectified by each member of a rescue corps reserving for himself an apparatus fitted only to himself.

Fig. XXX.—Diagrammatic Figure of the "WEG" Apparatus.



A=Oxygen cylinders; *B*=Oxygen valves; *C*=Oxygen connecting-pipes; *D*=Spare oxygen pipe; *E*=Lung governing arrangement; *F*=Padded leather cap; *G*=Oxygen supply pipe; *H*=Mouth-piece; *J*=Exhaling valve; *K*=Pipe through helmet to purifier; *L*=Purifier; *M*=Pipes to regenerated air-bag; *N*=Regenerated air-bag; *P*=Pipes through helmet to inhaling valve; *R*=Inhaling valve; *S*=Pressure gauge; *U*=Unions.

The jacket having been put on and the helmet-mask adjusted, the different parts of the apparatus will be found to occupy the following respective positions on the body:—

(1) **On front of body.**—On the head is the helmet-mask, the top of the helmet being composed of a padded leather cap. Over this from the mouth-piece runs the rigid tube conveying the expired air to the purifier, and in the reverse direction, a similar tube carrying the purified air to the mouth. In front of the mouth-piece and running downwards to the oxygen cylinder on the right side is the oxygen-supply pipe, from which the wearer may take, on emergency, a supply of pure oxygen.

Under the right arm-pit is placed one cylinder with the connected supply-pipe, and on the left side is the other cylinder with the pressure gauge.

(2) **On the back.**—Down the back of the helmet run the two tubes already mentioned, to a point where they separate into two separate tubes, one of which, carrying the expired air, passes to the purifying chamber, through which, after purification, the air enters the regenerated air-chamber by means of two tubes. From the upper part of this air-chamber a tube leaves, into which oxygen from the cylinders enters and mingles with the air coming from that chamber purified, and passing upwards over the helmet, opens by means of a valve close to the mouth of the wearer.

The entire apparatus weighs a little over 30 lbs.

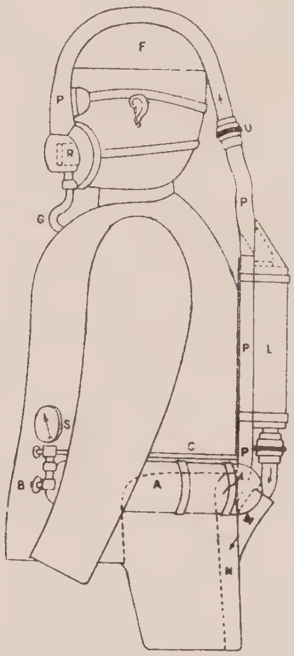


Fig. XXXI.—WEG Apparatus. For description of parts see Fig. XXX.

Second Group.—Pneumatogen Apparatus.

This apparatus differs from those in the former group in that the oxygen does not exist in the form of compressed gas in a cylinder, but exists potentially in certain chemical preparations, the peroxides of potassium and sodium, from which it is liberated by the action of the CO_2 and watery vapour given off in the expired air of the wearer. By the action of the former carbonates of potash and soda are formed, and of the latter, caustic potash and caustic soda, as shown in the following equations, viz. :—

1. $K\ NaO_3 + H_2O = KHO + NaHO + O_2$.
2. $CO_2 + KHO + NaHO = NaKCO_3 + H_2O$.
3. $CO^2 + KNa\ O_3 = NaKCO_3 + O_2$.

As is the amount of the CO_2 and the watery vapour exhaled from the lungs of the wearer, therefore, so is the amount of oxygen which is freshly liberated for the new needs of the wearer; in other words, the wearer makes his own oxygen: and as is the amount of his physical exertion, so are the respective amounts of CO_2 and watery vapour he gives off, and correspondingly, so is the quantity of liberated

PNEUMATOGEN APPARATUS.

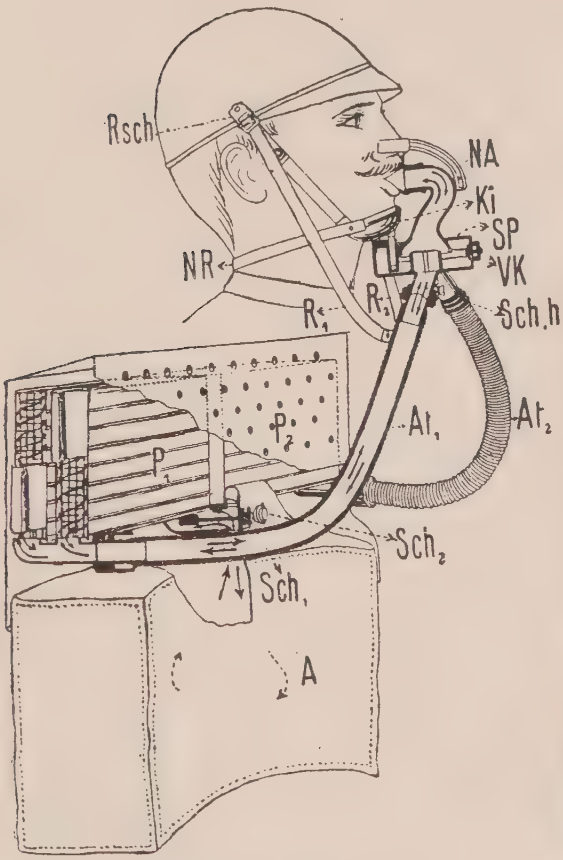


Fig. XXXII.

Fig. XXXIII.

Fig. XXXII.—Diagrammatic Arrangement of Parts.—*NA* = Nose-clip; *Ki* = China support; *SP* = Saliva trap; *VK* = Lock lid; *Sch.h.* = Switch cock; $At_1 At_2$ = Breathing tubes; *R*, *R* = Head straps; *Sch*, *Sch* = Screw-bolts; $P_1 P_2$ = Cartridges; *NR* = Neck straps; *Rsch* = Strap buckle; *A* = Breathing bag.

Fig. XXXIII.—Apparatus as adjusted to wearer. Description of parts of the apparatus may be discovered by reference to Fig. XXXII.

oxygen available for his use. This potassium-sodium-peroxide, $KNaO_3$, is much more portable than the comparatively heavy cylinders of the compressed-gas apparatus; besides, it can be put up in less bulk. Eleven of the cartridges, each containing 250 grammes of peroxide, with their cases and filters, weigh the same as two of the cylinders commonly used. The theoretical yield of oxygen from these eleven cartridges is 598 litres

or 21 cubic feet, and the actual yield of the gas from the cylinders from 170 to 274 litres or 6·0 to 9·7 cubic feet. Each cartridge latterly used in this apparatus contains 330 grammes of peroxide, and the theoretical yield of oxygen therefrom about 78 litres. It would not, however, be correct to assume that that yield is actually obtained, for apart from possible impurities in the peroxide itself, it is by no means unlikely that the products of respiration will fail to liberate all the available oxygen, owing chiefly to the failure of these to penetrate the mass of the cartridge substance.

As a matter of fact, experiments have proved that a "considerable amount of peroxide is found undecomposed. . . . It would appear, indeed, that in actual practice scarcely one-half of the theoretical quantity is obtained for use, though under very favourable circumstances, that is, very slow use, the proportion obtained may rise to about three-fifths." [1]

Experience has shown that after the products of respiration have acted upon this material, physical changes as well as chemical have also taken place. The granular material has become altered into a more or less caked mass, and this prevents further action of the watery vapour on the unaltered peroxide enclosed within, which, therefore, is useless for oxygen-generating purposes. Besides, in this apparatus the only forces at work in propelling the expired air through the absorbing medium are those which proceed from the respiratory action of the wearer.

Two types of apparatus have been devised, viz. :—Type 1, for self-rescue, and Type 2, for the use of rescuers of others.

We shall first consider the construction of the latter type. This form is strongly and well made. Its essential parts are as follows :—(1) Mouth-piece and tube; (2) Absorbing cartridges charged with mixed peroxides; (3) Tube between absorbers and breathing-chamber; and (4) Breathing-chamber. The mouth-piece is considered to be very suitable, as it leaks only if and when the mouth is much distorted. The nose-clip is efficient, and is declared by wearers to be comfortable. The mouth-piece, however, lacks teeth-pieces to be held in the teeth. From the mouth-piece a tube extends to the chambers containing the absorbing cartridges charged with the peroxides. While this chamber is filled with three cartridges, two only are at first put into action, the third being held in reserve for later action, if required. After the expired air has passed through this chamber, it passes into the breathing-chamber by the connecting tubes. This breathing-chamber always contains a supply of air, from which the wearer draws his supply, which is freshened by the new oxygen liberated.

The total weight of the apparatus is said to be about 8 lbs., but in the apparatus tested for the Mines Commission the weight was 14·5 lbs. The apparatus is calculated to serve the needs of the wearer for 80 minutes.

[1] *Report Roy. Com. on Mines*, First Report, p. 18.

Type 1 is of simpler form. It consists of the mouth-piece and tube as in the preceding type, but in this the cartridge-chamber is surrounded by the breathing-chamber. This type enables the wearer to stay in an irrespirable atmosphere for about one and a half hours in a quiescent condition, or to walk quietly about for half an hour, covering about one and a quarter miles in that time. It does not, however, enable the wearer to make severe exertion for any long period. Experiments made with this type of apparatus indicate that it is well fitted to serve for the purpose above indicated, and would permit a wearer to escape from immediate danger from the working-face to the pit-shaft.

It weighs between three and a half and five pounds.

There is some doubt, however, whether either form of this apparatus is now manufactured.

Third Group.

So far as we have been able to ascertain, the only form of apparatus falling within this group is the "Aerolith" apparatus.

In this apparatus the air itself is carried in liquid form in an insulated box loosely packed with asbestos. The air evaporating from the liquid material is the means of supply to the wearer of the apparatus.

There are no valves in this apparatus, as in the previous forms, to separate the expired from the inspired air. The expired air in this case is conveyed through a wide tube passing through the box containing the liquid air into a double bag behind the wearer, whence it escapes by means of a valve into the surrounding atmosphere. From the box which holds the liquid air the evaporated air leaves by means of a tube, which enters the wide expiratory tube near the mouth of the user.

The full charge of liquid air in the box amounts to five litres. Assuming this air to be of average quality, this charge ought to give off about 1000 litres of oxygen and 2000 litres of nitrogen, which quantities ought to be sufficient to supply the user amply for not less than one and a half consecutive hours. Experience has proved that one litre of liquid air takes about half an hour to evaporate.

The reason why, in the form of apparatus as devised, the wide tube to carry off the expired air is made to pass through the box containing the liquid air is, that the warmth of the expired air at a temperature of about 100°F., assists the evaporation of the air from its liquid to the gaseous form, and if this be borne out in experience, then the greater the exertion on the part of the user the more freely will the air be vaporised. One of the defects of the first apparatus was the absence of some means of signalising danger to the wearer from diminishing liquid air: indeed, the time-limit of safety during which the apparatus may be safely used in an irrespirable atmosphere could only be gauged by the decreasing

weight of the apparatus. This is a frail indicator to depend upon in such an instrument as this, since the time of greatest danger to the wearer is that point when, tired from his exertions, the wearer is least capable of distinguishing diminution in weight. The later model of instrument, however, is provided with a small clock, so that the wearer, knowing how long the carried amount of liquid air will take to vaporise, may take timeous warning to desist work and bear himself to safety.

Fig. XXXIV.
AEROLITH APPARATUS.

The other principal objection which has been urged against this type of apparatus is the difficulty to obtain easily supplies of liquid air. This objection has some force at the present time, but, doubtless, it will be remedied. To a certain extent, however, the same argument might be applied regarding the supply of oxygen in cylinders, but the objection does not go nearly so far, since oxygen in gas-tight cylinders will keep for almost any length of time, whereas the same cannot be said of liquid air.

On the other hand, there are two features of this type of apparatus which go a long way towards its recommendation and adoption, viz. :—(*a*) lightness in point of weight, and (*b*) comfort in use. Besides, when the supply of liquid air is standardised to last a definite period of time, set down in minutes, and irrespective of the severest amount of exertion on the part of the user, the installation of a visible clock should further recommend it for rescue operations. Further, the absence of a cycle of movement of respired air within the apparatus is an excellent feature, and there is no need, therefore, to carry absorbing material, because the expired air with its products are thrown off into the surrounding atmosphere at the moment of exhalation.

According to experiments which have been made while the apparatus was being worn by men, the oxygen supply was always found to be

ample, the percentage being within the limits of 40 and 75 per cent. It was also noted that if the wearer continued to wear the apparatus after the liquid air had been vaporised and until the breathing-bag was quite empty, the air left within the apparatus contained even then about 50 per cent. of oxygen. During the currency of the experiments, the amount of CO_2 gas found in the early stages varied between 0·2 and 2·5 per cent., but in the later stages it rose to between 3·5 and 5·4 per cent. in the breathing-bag.

These results seem to point to the likelihood that some of the expired air not only did not escape from the breathing-bag, but that it was partly taken back into the air of inspiration. The experiment also showed that the chief difficulty in breathing arose from excess of fresh vaporised air coming from the liquid air-box which, in turn, produced hyperdistension of the entire apparatus, with the result that some difficulty was experienced by the wearer on expiration. It was considered that this trouble arose partly from the accumulation of frozen watery vapour in the expiratory tube where it passed through the liquid air-box, partly from insufficiency of size of the valve of outlet of the expired air into the surrounding atmosphere, and partly also from the smallness of the tube connecting the two breathing-bags which, externally, look as one bag. The remedies which have been suggested to obviate this discomfort to the wearer are (1) to introduce a blow-off valve on the expiratory tube, and (2) to replace the present mouth-piece by a mask.

The reader will, perhaps, be able to form a better conception of the apparatus from the following description. From the mouth-piece or mask passes a wide tube, whose duty is to carry away the expired breath of the user. This extends down through the box containing the liquid air, and ends in the first breathing-bag at its bottom, when the air rising in the bag passes through an opening which communicates with the top of the second breathing-bag, and then passing downwards through this bag, escapes by the expiratory valve into the surrounding atmosphere. The fresh air, vaporised from the liquid air, passes to the mouth of the wearer in the following manner; from the top of the box of liquid air comes a tube which, running parallel to the expiratory tube already named, opens into this tube a short distance from the mouth of the wearer, and thus yields a supply of fresh air. Placed on the top of the liquid air-box is a small clock, by which the time of use of the apparatus can be noted by the user, and warning is thus given to keep well within the necessary margin of safety.[1]

[1] Simonis: *Trans. Inst. Mining Eng.*, December 1906.

Comparative Uses and Values of Apparatus for Rescue Work.

1. **Apparatus used by rescuers.**—Having dealt with the different forms of apparatus available for the use of rescuers who may be compelled to undertake upon occasion more or less hard and hazardous work during a limited length of time, it will be sufficient to indicate their comparative differences with respect to certain important particulars.

It will have been noted that the mode of supply of oxygen differs somewhat in these. In the Esseff, the Draeger, the Proto-Fleuss, and the WEG, the supply is derived from the cylinders of the compressed-gas, but that whereas in the first three of these the supply is constant, in the last-named it may be made variable according to the needs of the user. One advantage of the former mode of supply is, that it leaves the user free to do the work before him without the need to consider the adjustment of valves. Besides, the constant supply of oxygen compels the continuous change of the air respired, and during any short interval of comparative rest the sweep of oxygen frees the apparatus of any excess of nitrogen which may be present. The important point, however, in connection with any apparatus professing to supply a continuous ample stream of oxygen is, that the apparatus should be set so that it shall deliver between one and three-quarters and two litres of oxygen per minute.

At the same time, the arrangement in the WEG apparatus permits the user to save his oxygen when occasion permits, even to cutting it entirely off, should he chance to get into ordinary respirable air, as well as to inhale pure oxygen, if necessary, to revive himself.

While a gauge is supplied with all such forms to indicate the progress of the diminishing pressure in the oxygen cylinders, it would be better if the gauge-pressure were calibrated to time.

In the other forms in which the oxygen is not supplied under pressure, consideration must chiefly be given to the efficiency with which they yield their oxygen. It is doubtful if the Pneumatogen apparatus is as yet so perfected as to give certainty in this particular, owing to the clogging produced in the peroxides which yield the oxygen by the action thereon chiefly of the watery vapour of the expired breath.

To the Aerolith apparatus the objections offered do not so directly apply, since the medium of respiration is provided in the form of liquid air. The advantage of this apparatus is that there is no call for the provision of absorbents of the CO_2 and watery vapour of the expired air, as these are permitted to escape into the surrounding atmosphere. At the same time, the watery vapour is liable to be condensed into the form of ice within the expiratory tube in its passage through the liquid air-box, owing to the frigidity of temperature of the liquid air.

In all the other forms of apparatus absorbents of the products in the expired air are essential, hence the mode of absorption and its sufficiency

become of importance. There can be little doubt that the best form of absorbent is that which will take up a fairly large amount of watery vapour without the material becoming damp and pasty. A large superficies of granular material is, probably, the best from that point of view, but on the other hand, such a form demands large cartridge-space and tends to make the entire apparatus bulky and ponderous. It has been said that, in respect of ineffectiveness of absorption, the "Shamrock" apparatus might be improved with advantage, and probably that might be achieved by a change in the nature of the absorbent used.

The remaining condition to be considered from the practical point of view is the weight of the apparatus. It will have been observed that in this respect the apparatus differ. These differences depend on whether one or two cylinders and whether the mouth-piece arrangement or the helmet are used. But with the exception of the Aerolith, which weighs about 22 lbs , the others weigh something over 30 lbs.

There can be little doubt that the effectiveness of any of the forms of apparatus named will depend in great measure (1) on the selection of the men who are to use them, (2) on the kind and amount of training in the use of the apparatus which the men have received, and (3) on the familiarity of the men with their own individual apparatus.

The following table, taken from the First Report of the Royal Commission on Mines, gives the conclusions arrived at after examination of and the tests made with the forms of apparatus which have been discussed.

TABLE XXIII.[1]

Apparatus.	Weight in lbs.	Oxygen Capacity in Litres and Cubic Feet.	Removal of CO_2.	Supply of Oxygen.	Air Tightness.	Comfort and Convenience.
Pneumatogen (Type II.,	14½	163*(5·8)	Good	Poor	Good	Very Good
Draeger . .	36-39	251 (8·9)	Good	Good	Good	Bad
Shamrock .	36	274 (9·7)	Bad	Good	Good	Good
Fleuss . .	31	228 (8·0)	Good	Good	Good	Good
WEG . .	30 +	150 (5·3)	Good	Good	Good	Good
Aerolith .	22	?	Fair	Good	Good	Very Good

* Maximum theoretical yield: 80 to 100 litres is the more probable figure in actual practice.

[1] *Roy. Com. on Mines*, First Report, p. 42.

Restoration of the Apparently-Asphyxiated.

The simplest methods of trying to restore animation to an apparently-asphyxiated person are (1) artificial respiration, and (2) the administration of oxygen, coupled with the application of warmth to the body or placing the unconscious person in a warm room. It is not necessary, perhaps, to name, much less discuss, the different modes of inducing respiration by artificial means, since these form part of first-aid teaching, and may, therefore, be presumed to be familiar to those who at mines are charged with the restoration and first-aid treatment of injured persons.

When intelligently administered, the exhibition of oxygen for inhalation to persons whose respiratory function continues, even though difficult or irregular, is of the first importance. It is, therefore, desirable that the proper mode of its use should be understood. The oxygen will be of little use to the patient, for example, if the source of supply is held some inches from the face, and not of much more use even if conveyed to the mouth and nostrils by means of a funnel held over the face, for the simple reason that the major portion of the gas escapes into the surrounding atmosphere. On the other hand, the danger zone is entered if the gas is administered at too high pressure, as inflammation of the lungs may thus be produced. Several observers, among others, Lorraine Smith, Macleod, and Hill, have induced such lung inflammation experimentally in animals to whom oxygen at high pressure had been administered for a length of time.

The best results seem to be obtained when the gas is administered under partial pressure, that is, higher than normal atmospheric pressure; in short, when the atmosphere which the patient has to breathe is hyper-oxygenated.

Two things are essential, therefore, for this purpose, viz. :—(*a*) a mask, made of some non-absorbent material, which may be applied more or less accurately and closely to the face of the patient, so as to prevent the escape of the gas, and (*b*) the admission of the gas from the cylinder into the mask under such degree of pressure as will produce a definite feeling of coolness when applied to the face. By practice and experience the required degree of pressure may be judged by the intensity of the hissing sound produced by the escaping gas from the pressure-gauge of the cylinder. In ordinary cases, where the impending asphyxia has been produced by the inhalation of noxious gases, there is no need, perhaps, as in cases where the oxygen is being administered for lung diseases, to warm the gas before it is inhaled.

The application of warmth to the body in cases of this kind is of prime importance, for the reason that the bodily temperature is likely to be sub-normal or to incline in that direction.

Since, according to the Statutory Rules and Orders, 1913, oxygen reviving apparatus must be kept at mines, it is important that such apparatus should always be kept in readiness and in good order for immediate use.

The Pulmotor.

An ingenious apparatus has also been devised for the purpose of resuscitating persons who have been rendered more or less asphyxiated in

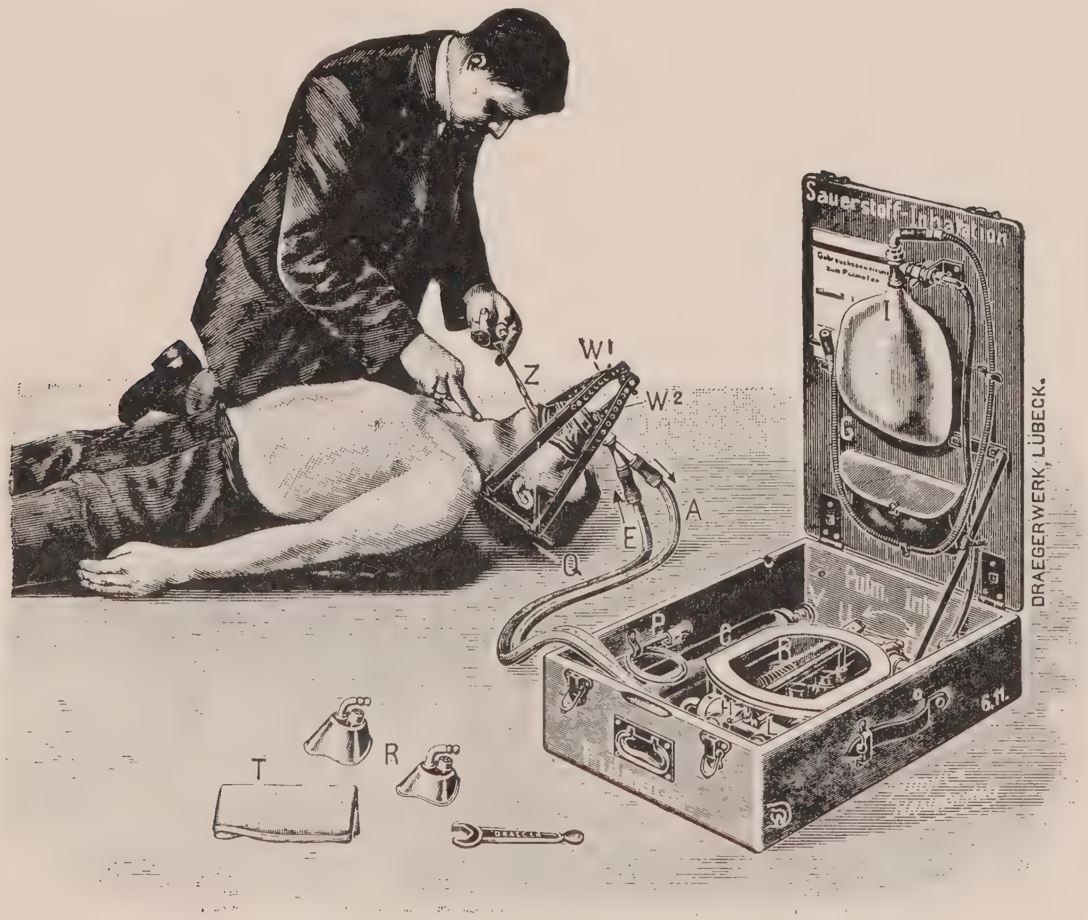


Fig. XXXV.—The Draeger Pulmotor. (For description, see text.)

an irrespirable atmosphere, and whose unconscious bodies have been removed from their deadly position. This is an invention of the Draeger Company, and, perhaps, of others, and has received the name of the Pulmotor.

The Pulmotor may be employed for artificial respiration, or simply as a means of administering oxygen to such persons. It may be employed in all circumstances in which asphyxia is jeopardising the life of a person from the presence of any liquid or gaseous medium in the air-passages

and lungs. It may thus prove serviceable in threatened asphyxiation by inhalation of toxic gases and fumes, in apparently-drowned persons, in persons under the influence of electric shock, and in other conditions such as suspension of respiration while under the influence of anæsthetics. Before it may be used in apparently-drowned persons, however, the air-passages and lungs must be emptied, as far as possible, of their contained water.

The same apparatus may be used merely to administer oxygen in cases in which the use of that gas may be reckoned to operate beneficially as an aid toward recovery.

Description of the Pulmotor.—The entire apparatus is housed in a handy wooden case, the weight of the whole being about 46 lbs. The apparatus is partly fixed to the walls of the case. When it is about to be used, therefore, the lid of the case must be turned well back on its hinges. The apparatus will be found to consist of two separate parts, viz. :—(1) an oxygen-inhalation installation, which is mounted on the lid, and (2) the special apparatus for artificial respiration, which is housed in the case itself.

Both have in common the oxygen cylinder, *C*, and the pressure-reducing valve, *D*. Either apparatus may be set in operation separately by turning the lever, *U*, to the right or left on the reducing valve. When the lever is turned to the left, the oxygen passes to the pulmotor, when turned to the right, toward the oxygen-inhalation apparatus. The oxygen cylinder, *C*, is closed by the valve, *V*, and as soon as this valve is opened, one or other of the apparatus, depending upon the use to which it is to be put, begins to work. The cylinder contains $11\frac{1}{2}$ cubic feet of gas, and when a full cylinder is started in the process of artificial respiration it will continue in operation for 40 minutes consecutively. The oxygen passes from the reducing valve to the injector, *S*, which has the power of drawing in a large volume of air with a certain degree of suction, and of propelling that air forward with equal force through the flexible tube in front of the injector. It is this suction and this delivery which serve as the motive power of the apparatus, alternately filling the lungs by pressure and emptying them by suction, no injury being inflicted during the action. The pressure in the pulmotor is equivalent to eight inches of a water-gauge, and the suction to a pressure of ten inches.

The important part of this apparatus for artificial respiration, now being considered, is what is called the *automatic reversal*. In Fig. XXXVI. will be seen a small accordion-like bellows. It is this which effects the automatic reversal of the apparatus from suction to delivery and *vice versâ* continuously. This bellows is connected with the air-tubes. During inflation, the same pressure exists in the bellows as in the lungs of the person, and as soon as the latter are filled, the bellows becomes inflated,

and in moving forward causes the valve to be reversed automatically into position for suction. The operation is thus reversed; and as soon as the lungs have been emptied by expiration, the bellows contracts and in turn automatically reverses the valve again into position for inflation or delivery: and so on *da capo*. Hence it follows that the rhythmic action of the pulmotor readily adapts itself to the lung capacity in each case; thus the rhythm will be slower when the lungs are seldomer filled in persons whose lung capacity is large, and quicker when the lungs are more frequently emptied because their capacity is smaller.

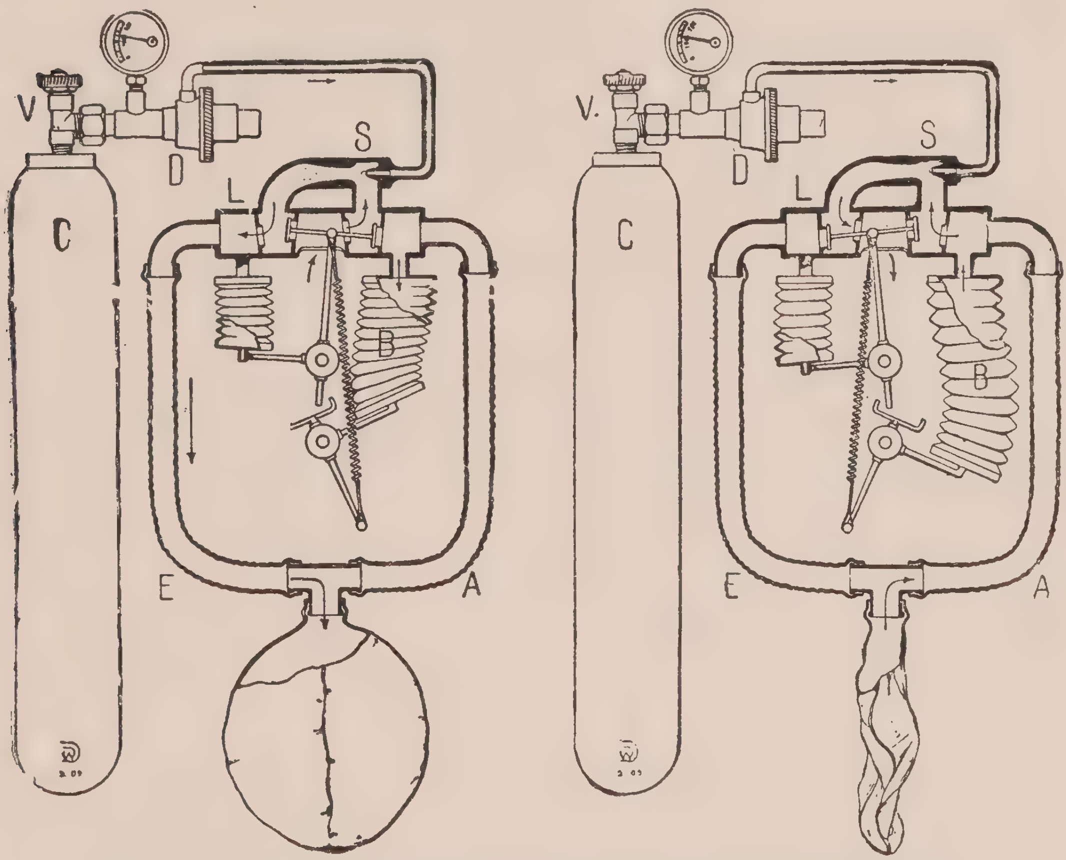


Fig. XXXVI.—Automatic-reversal mechanism of Pulmotor.

C=oxygen cylinder; V=reducing valve; S=injector; L=air-reversing chamber; B=leather-accordion bellows; E=tube for pure air; A=tube for exhaled air. In the Fig. on the left the bag is shown collapsed, and that on the right inflated, these being intended to show the automatic action of the apparatus in deflating and inflating the lungs.

The apparatus, therefore, performs the functions of inspiration and expiration without any assistance being called for from the hands of the operator, whose attention, however, must be directed to keeping the wind-pipe open and the gullet closed.

To the mask to be fitted around the nose and mouth of the unconscious person, two flexible breathing tubes must be connected. One of these,

E, serves exclusively for the supply of pure oxygen and air, while the other, *A*, is used entirely for drawing off the expired air. By this arrangement no vitiated air is able to return to the lungs, as might happen if a single tube only were employed. It is essential that the mask be fitted air-tight. It is secured to the face by two pairs of straps, which branch from a padded ring placed under the back of the head. This is accomplished by a little manipulation. Before adjusting the mask, however, the operator must make certain that the tongue does not obstruct the passage of air into the lungs. This may always be assured by catching the tongue with a pair of forceps, *Z*, provided with the apparatus, and pulling the organ well forward to the edge of the mouth. This may require, in some cases, the use of some little force, which should be exercised with care. But when this is done, and the mask has been adjusted air-tight around the mouth and nose, the forceps continuing to maintain the tongue in the desired position, the apparatus is ready to be started, provided that pressure by the finger, sufficient to occlude the gullet passage, is now made on the prominent part of the larynx or voice-box, for if this be not done, the oxygen and air, which are being passed into the mouth and nose from the apparatus, may find their way into the stomach, where they would be of no use for breathing purposes.

On the lid of the case, there will be observed in Fig. xxxv. a special flexible metal tube, *G*, and two masks, *R*, one of the latter being for males and one for females, and by one of which the oxygen is exhibited. The lever for the administration of oxygen must be turned to the right side, when the apparatus will yield a supply of 244 cubic inches or four litres of the gas per minute, this being taken by the person from the bag, *I*.

Practical Rescue and Aid Operations in Mines.

It does not fall within the scope of this book to discuss in any detail the practical working of rescue operations in pits and mines: that must be left to those who are more conversant with all the circumstances of mining operations. At the same time, there are aspects of rescue work, involving questions of medico-legal interest, which are apt to be raised in legal inquiries, and of risks to the lives of rescuers which fall within our province.

Since rescue and aid work mainly follows, perhaps, the occurrence of more or less serious mine accidents, such as an underground fire or an explosion of fire-damp, in both of which toxic gases are generated which rapidly jeopardise the lives of men below ground, there are at times less serious occurrences, such as sudden outbursts of gas, etc., which equally and not less promptly call for the work of rescue.

So important are these aspects of the subject that a special committee, called the Mines (Rescue and Aid) Committee, was appointed in 1910 by the Secretary of State for the Home Department to consider and report on the matter.[1]

As the result of their labours, a Draft Order was recommended by the Committee, the chief points of which only may now be considered. These were as follows :—

1. That the Order should supply to all mines in which coal is worked, except in mines in which the total number of employees is less than 100, and in which, in the opinion of the Secretary of State, the organisation of a Central Rescue Station from which these could be served, was impracticable, and then only by the exemption of the Secretary of State.

2. No person, unless authorised by the manager, or official appointed by the manager for the purpose, should be allowed to enter a mine after an explosion of fire-damp or the occurrence of a fire, for the purpose of engaging in rescue work.

3. At every mine, except an exempted mine, there should be organised and maintained, as soon as is reasonably practicable, competent rescue brigades on the following scale, viz. :—(*a*) where the number of underground employees is less than 250, one brigade; (*b*) where the number is more than 250 and less than 500, two brigades; (*c*) where more than 500 and less than 800, three brigades; and (*d*) where more than 800, four brigades.

If, however, the owner, agent, or manager of a mine having fewer than 100 underground employees has acquired the privilege of calling a brigade from a Central Rescue Station, he should be deemed to have complied with this provision.

A rescue brigade should consist of not less than five persons employed at the mine, who shall have been selected because of their underground knowledge, coolness, and powers of endurance, and shall have been certified medically fit, the majority of whom shall hold a Certificate of the St John's or the St Andrew's Ambulance Association, shall have received instruction in reading mine plans, in the use and construction of breathing apparatus, in the properties and detection of poisonous or inflammable gases, and in appliances used in connection with mine rescue and recovery work. One of the brigade should be selected as leader, who should act as captain. A brigade should not be deemed competent unless it has undergone a systematic course of training approved by the Secretary of State, and every such brigade, after the preliminary course of training and instruction, should undergo one day's training at least in every quarter; and arrangements should be made at every mine

[1] *Report of Departmental Committee on Organisation of Rescue and Aid in Accidents in Mines*, 1911.

for summoning the rescue brigade immediately their services are required.

4. If it can be clearly proved that the necessary number of persons will not consent to form such brigades, or having offered, fail to be trained or to maintain their training, the owner, agent, or manager of a mine should not be in any way penalised, provided that he has endeavoured to the best of his ability to constitute the requisite brigade or brigades, and has afforded every opportunity to the workers to undergo the necessary training, and has under these circumstances made a *bonâ fide* attempt to arrange for the supply from a Central Rescue Station of such rescue brigades as he is unable to provide at his mine.

5. There should be provided and maintained at every mine, sets of portable breathing apparatus in the proportion of two sets to each brigade required in Section 3 (*a*), said apparatus to be capable of enabling the wearer to remain for at least one hour in an irrespirable atmosphere, and to be kept ready for immediate use. The apparatus must be housed in suitable receptacles in a dry and cool room.

If an owner, agent, or manager of a mine has acquired the privilege of calling for such appliances from a Central Rescue Station, he should be deemed to have complied with this requirement, provided that said Central Rescue Station is not more than ten miles distant by road from the mine and is in telephonic communication with the mine.

If at the date of this Order becoming law it can be shown that it is not possible to procure said breathing apparatus, owing to lack of supply, the owner, agent, or manager shall be deemed to have complied with the Order if he procures such appliances as soon as is reasonably practicable.

There should also be provided and maintained at every mine :—(*a*) two or more small birds or mice for testing for carbon monoxide ; (*b*) two electric hand-lamps for each brigade, ready for immediate use and capable of giving light for at least four hours ; (*c*) a safety lamp for each member of the rescue brigade for testing for fire-damp ; and (*d*) tracings of the workings to be kept up-to-date, not more than three months previously, showing the ventilation and all doors, stoppings, and air-crossings and regulators, and distinguishing the intake-air by a different colour from the return-air, which plans shall be in suitable form for use by the brigade.

6. In every Central Rescue Station there should be kept and maintained not less than fifteen sets of breathing apparatus, with means of supplying oxygen or liquid air, to enable such apparatus to be constantly used for two days, and of charging such apparatus, and twenty hand-lamps, four oxygen-reviving apparatus, ambulance boxes, such as are provided by the afore-named Ambulance Associations, together with antiseptic solution and fresh drinking-water, and cages of birds and mice. A motor car should be kept in constant readiness.

7. Every Central Rescue Station shall be placed under the immediate control of a competent person conversant with the use of the appliances.

8. At every mine there should be rules for the conduct and guidance of persons employed in rescue work in or about such mine as may appear best calculated for the carrying out of rescue operations.

9. The expression "Central Rescue Station" means a station situated centrally to serve several collieries.

By the powers conferred upon him by Section 86 of the Coal Mines Act, 1911, the Secretary of State made General Regulations, dated 10th July 1913, and which came into force two months after their publication in the *London Gazette* of 15th July of that year, of which the sections laid down in the foregoing Draft Order of the Departmental Committee, with some alterations, formed part. These Regulations are known as "Statutory Rules and Orders, 1913, No. 748," and the above sections of the Draft Order correspond to Regulations 138 to 146 inclusive of Part IV., which is headed "Rescue and Ambulance."

In order to give the reader information relative to the changes between the Order of the Departmental Committee as drafted, and the Regulations as issued in the form of a Statutory Order, it will only be necessary to compare each section of the former with the corresponding Regulation of the latter, and to draw attention to any changes in them.

Regulation 138, corresponding to Section 1, is the same. Regulation 139, corresponding to Section 2, in regard to the authority at a mine who is capable of giving admission to enter a mine after an explosion or a fire, contains the following: "or, in the absence of the manager or such official, by the principal official of the mine present at the surface." Regulation 140, corresponding to Section 3, contains the following alterations respecting the number of rescue brigades relative to the number of underground employees, viz.:—"where the number of underground employees is 250 or less, one brigade; where more than 250 but not more than 700, two brigades; where more than 700 but not more than 1000, three brigades; and where more than 1000 employees, four brigades"; moreover, the following has been added:—"A group of mines belonging to the same owner, of which all the shafts or exits for the time being in use in working the mines lie within a circle having a radius of two miles shall, for the purpose of ascertaining the number of brigades required, be treated as one mine." The remainder of the Regulation is the same as the section in the Draft Order, except that with regard to the training to be given to the men constituting a competent rescue brigade, two of the practices which are required to be carried out on one day in each quarter shall be given in the mine itself. Regulation 141, or Section 4, remains the same. Regulation 142, or Section 5, remains the same for the most part, but the proviso in the Draft Order,

relating to the possibility of procuring breathing appliances, is omitted in the Regulation, and it is ordained that at every mine, in addition to the appliances named in the Draft Order, "one oxygen-reviving apparatus" and "an ambulance box" shall also be provided and maintained. Regulation 143, or section 6, is exactly the same, except that "mice" do not require to be kept for the detection of carbon monoxide at a Central Rescue Station. Regulations 144, 145, and 146, corresponding to sections 7, 8, and 9 are the same.

Under part IV. of the Regulations, certain requirements for ambulance assistance have been added. These constitute Regulations 147, 148, and 149. Regulation 147 ordains that in every mine there shall be provided and kept in good order and ready for immediate use at a convenient spot *in the district of each fireman, examiner, or deputy*, and also at the mine or other convenient place on the surface, (*a*) a suitably-constructed stretcher, and (*b*) a box containing a sufficient supply of suitable splints and bandages, adhesive plaster, boric vaseline, cotton wool and tincture of iodine, or other suitable antiseptic solution. For the purpose of this Regulation adjoining districts may be treated as one district, provided the number of persons employed does not exceed 50. These requirements shall not apply, however, to any mine, seam, or district of a mine in which dampness would make it impossible to keep the appliances in good order. When a dispute arises between a manager and his workmen as to such possibility, the matter shall be referred to the inspector of the division, who shall have power to decide the same. The manager or other qualified official shall personally inspect the foregoing appliances once a month, and satisfy himself that they conform to the above requirements.

Regulation 148 ordains that in every mine, other than a small mine, the manager shall arrange, if possible, that there shall be at least one man trained in first-aid and holding a certificate of the St. John, the St. Andrew's, or other Association, in the district of each fireman, examiner, or deputy at any time when twenty persons or more are being employed in the district. If less than twenty persons are employed in each district the manager shall arrange, if possible, that there shall be below ground during each shift at least one man so trained and having the certificate as aforesaid.

This Regulation shall not come into force until 1st April, 1914.

Regulation 149 ordains that there shall be provided and kept in good condition at every mine a suitably-constructed ambulance carriage. But this requirement shall not apply :—(1) to any mine having fewer than 100 employees, if in the opinion of the Secretary of State it is so situated that it cannot be served from a Central Rescue Station, hospital, or other place, or by arrangement with other mines for the joint provision of

a carriage; (2) to any mine in which the number of workers is less than 500, if the owner, manager, or agent has acquired the privilege of obtaining the use of such a conveyance when required from any place distant not more than ten miles from the mine and in telephonic communication with the mine; (3) to any mine having 500 employees or over, if the owner, agent, or manager has acquired the right as in (2), and the inspector of the division is satisfied that the arrangements are such as to ensure the prompt attendance of the carriage at the mine.

A group of mines belonging to the same owner, or to owners who have entered into an arrangement for the joint provision of an ambulance carriage, shall for the purpose of this Regulation be treated as one mine, if all the shafts or exits for the time being in use in working the mines, lie within a circle having a radius of not more than two miles, or where a motor ambulance carriage is provided in constant readiness and facilities exist at each mine for summoning the carriage by telephone, within five miles. (For later modifications, *see* Appendix.)

Much has been written in the Mining press concerning the best methods of recovering a coal mine after explosions and fires, but one of the best guides, so it appears to us, on the subject is that from the pen of Sir W. E. Garforth which, based as it is upon practical experience, is therefore of the greater value.[1]

Having constructed an experimental gallery in which practice in the use of breathing apparatus might be obtained, this writer is entitled to write with some authority. He has very properly insisted on the need for such practice in the use of rescue apparatus in the following decisive words:—"That unless the wearer of the apparatus has systematically and regularly practised for three months in a gallery on the surface made like the damaged roadway of a mine, with confined spaces, etc., and been surrounded with an irrespirable, hot, and occasionally humid atmosphere, for at least two consecutive hours, then such an apparatus, instead of being a help to the wearer, may prove to be a *Death-Trap*. On the other hand, the writer believes that with proper training a brave and cautious man, accustomed to underground work, will be able to render valuable service in saving life, affording relief to sufferers, safeguarding explorers, and protecting property."[2]

It appears to us to be absolutely essential, in order that the best results in rescue and aid work may be achieved by the use of rescue apparatus, that the men who are to use them should be well trained in their use. This is alluded to in the Statutory Rules and Orders. There can be no doubt, in the absence of adequate training, that men run not

[1] Garforth: *Suggested Rules for Recovering Coal Mines after Explosions and Fires*, 1909.

[2] *Trans. Fed. Inst. Min. Eng.*, Vol. XXII., p. 179. *Loc. cit.*, p. 27.

inconsiderable risks when wearing them under those circumstances. Fatal accidents to rescuers using apparatus have had to be recorded from time to time. One of the German brigade, who came to help at the Courrières disaster, died. In Austria nine lives have thus been lost, and in the majority of these instances when using an apparatus in which the oxygen-supply came from chemical peroxides. A number of deaths have taken place also in this country. In 1906, at a fire at Hampstead colliery, where the Draeger and WEG apparatus were used, one of the members of the rescue brigade, in his anxiety to help, went further than his supply of oxygen would carry him in safety, and lost his life in consequence. At a recent disaster, moreover, one of a rescue brigade lost the mouth-piece of his apparatus, and inhaled some of the surrounding poisonous air. In the words of a witness "at once he got completely out of control, and went out of his mind," and when the other members of the rescue party replaced the tubes, he pulled them out again. Besides, fatal occurrences have happened during practice with the apparatus at rescue stations, owing to the apparatus being out of order, and for other reasons. While, however, the foregoing must be written, it cannot be gainsaid that such deplorable accidents are less likely to occur in the future among men who have been adequately trained.

Observations of value have been made regarding the physiological effects of wearing rescue apparatus, to which attention must be drawn. These have been made on men working in experimental galleries. One series of observations made by Fraser Harris calls for notice.[1]

The observations were made on healthy men in the experimental mine at Bournbrook, the ages of the men ranging from 26 to 50 years, their heights from 5 ft. 7 in. to 6 ft. 6 in., and their weights from 55 kilos to 89 kilos, or from nearly 122 lbs. to 197 lbs.

The apparatus employed in the tests were :—(*a*) Fleuss, (*b*) Draeger, and (*c*) Meco, with constant supply of compressed oxygen, and (*d*) WEG, with automatic oxygen supply, (*e*) Aerolith, in which liquid air is the source of oxygen supply, and (*f*) the Hall-Rees, in which the oxygen was derived from peroxides.

The types of apparatus used were :—(1) those with helmet covering the head and face, (2) those with half-mask covering nose and mouth only, and (3) those with mouth-piece only; smoke goggles to be worn with this type in smoke-laden atmospheres.

So far as the temperature of the place of experiment was concerned, the tests were divided into three groups, viz. :—(1) in the ordinary temperature of the mine; (2) in hot, moist, irrespirable (smoky) atmospheres, the wet-bulb temperature being between 80° F. and 85° F.; and (3) in hot, moist, but respirable atmospheres, the wet-bulb temperature

[1] *Brit. Med. Jour.*, Vol. II., 1911, p. 611.

being between 90° F. and 98° F. Before each test the men were weighed and their pulse and temperature taken, the same being done after the experiment.

From these experiments, Harris concluded that the helmet was objectionable for the following reasons, viz.:—(*a*) ventilation of the face was hindered, sweat was not evaporated, and the moisture dimmed the window of the helmet; (*b*) it prevented, therefore, free vision by the wearer of his surroundings either above his head or when crawling on his hands and knees; (*c*) it diminished the power of hearing; (*d*) it gives an artificially large "dead space" for breathing; and (*e*) when applied air-tight to the head in smoke-laden atmospheres, the pressure at contact parts becomes painful. With the mouth-piece form of appliance, plugging of the nostrils or compression by means of a nose-clip is uncomfortable, and on account of the rigidity of the tubes connected with it, and their weight, the mouth-piece is apt to be pulled from the mouth of the wearer when he is in a constrained position, more especially when stooping.

Effect on the pulse.—The action of the heart was accelerated in all the tests, the lowest increase noted being 16 beats, and the highest 58 beats per minute. In all cases, however, the heart had resumed its normal beat within fifteen to twenty minutes after the cessation of the test.

Effect on respiration.—Nothing further than physiological increase of breathing, or hyperpnœa, was observable, even at the end of two hours' hard work. No difficulty of breathing or dyspnœa was found.

Effect on bodily temperature.—The temperature of the wearers always showed a rise, which varied from 0·2°F. to 2·0°F. in tests in groups 1 and 2, but in those in group 3, when the wet-bulb temperature was 90°F. and higher, the rise amounted to between 2·5°F. and 2·8° F. Where the rise was not above 2°F., this was attributed to the profuse sweating experienced.

Effect on weight of men.—In every case, it was found that there had been some loss. The total loss varied much in different individual wearers; thus one man weighing 55·5 kilos or about 122 lbs. lost 500 grams, or 1·1 lbs. avoirdupois; another, weighing 88·6 kilos or about 196 lbs., lost 0·498 lb.; and another, weighing 67 kilos or 147·7 lbs., lost 1,250 grams or 2·75 lbs. As was to be expected, the largest loss of weight occurred in those tests made in an excessively hot and moist atmosphere.

Absorption of carbon dioxide and moisture.—In all the types of apparatus used, this gas was absorbed moist by means of sticks of NaHO. The weight of the gas and moisture was obtained by weighing the

"regenerators" or "absorbents" before and after each test. The results were summarised in the following table.

TABLE XXIV.

ANALYSES OF GASES IN THE APPARATUS.

Apparatus.	Incoming Air.			Outgoing Air.		
	Temp. F.	O_2 %	CO_2 %	Temp. F.	O_2 %	CO_2 %
Fleuss . . .	92°	74·0	0·57	96°	61·5	2·45
Draeger . .	74°	54·1	0·00	82°	68·0	1·50
Meco . . .	88°	55·8	0·00	89·5°	50·0	1·89

In certain individual analyses, however, the absorption of CO_2 was not so satisfactory, for in the incoming air in the Fleuss apparatus it was found occasionally to be as high as 8·75 per cent., and in the Draeger and Meco, 2·25 per cent. Harris is of opinion, which we share, that this gas in the air to be inspired ought not to exceed, if possible, one per cent.

As a result of the absorption of this gas and moisture from the expired air, an amount of heat was generated in the apparatus which came to be unpleasant to the wearers. It made the inspired air disagreeable or, indeed, actually painful to the throat, and in the case of the Fleuss apparatus the skin of the back and abdomen of the wearer became uncomfortably hot. Because of this heated condition of the air, pharyngeal catarrh, which sometimes persisted for more than a week, was produced ; not improbably this was aided by the undue moistness of the air.

One of the factors of considerable importance, relative to the ability of any wearer of a rescue apparatus in a mine to do hard work for any length of time, is the temperature and moisture of the air in which the work has to be done. When, for example, the temperature of the wet-bulb thermometer is over 85°F., the wearer is much more rapidly overcome by a feeling of tiredness and lassitude, and consequently is unable to perform work over a long period. Respecting the time which a wearer may be expected to work in a mine, therefore, attention ought for this reason to be directed to the temperature of the wet-bulb on every occasion.

Harris draws attention also to the risks of oxygen-deficiency occurring from some unexpected accident to the apparatus itself. Should such

happen, danger may arise to the person in such a plight, and for this reason it has most properly been urged that no single person or even two persons should be permitted to enter an irrespirable zone, but that a brigade, consisting of not fewer than five persons and under the direction of a leader, should work together.

The course of training at Howe Bridge Rescue Station has been described from personal experience by McElligott[1] as follows:—"The curriculum consists of two hours' work in galleries filled with smoke and sulphurous vapours; the galleries are such as one meets with in a coal mine, tunnels through which one had to travel either on all fours or in a crouching position. The floors are of hard, sharp flints; the roof supported by wooden props and bars. The work consists of filling and emptying a coal-tub (small wagon) with bricks; setting wooden props to support the roof; bringing out a mannequin representing a dead, or perhaps grievously-smitten man; fixing a brattice screen; and other jobs such as one would be called upon to do in case of fire in a coal mine. During the practice we breathed 80 or 90 per cent. of pure oxygen through the apparatus for the whole of the time. My personal fitness," continues the writer, "at the end of the two hours was one of extreme fitness. . . . During the six weeks or two months over which the course of instruction extended in weekly practices, I felt more fit than I had done for the previous six months. . . . I am convinced of the value of oxygen in rescue work."

In the equipment named in the Draft Order already considered, it will have been noted that the use of birds or mice in cages is enjoined. On this point it may be said that while, in general, such small animals are likely to give early notice of the presence in an atmosphere of toxic amounts of carbon monoxide, there are occasions on which they fail to do so. At the recent Senghenydd disaster, for example, it is recorded[2] that as late as 21st October an advance party of the repair gang were in serious danger from gas poisoning. One of the party carried a canary "which it was expected would give a warning, but before any signs of distress were noticed in the bird several of the party were overcome. With the assistance of the Aberavon Rescue Brigade wearing the Draeger apparatus, the party was rescued. The men were treated in the temporary hospital, where all recovered."

Range and Scope of Rescue Operations.

While doubtless rescue work will discover its chief value in such mine disasters as underground fires and explosions, it is likely in the future to play a more important part in the minor accidents by gassing which not

[1] *Brit. Med. Jour.* Vol. I., 1912, p. 159.
[2] *Brit. Med. Jour.* Vol. II., 1913, p. 1113.

infrequently occur in pits, not only in the rescue of men overcome by sudden outbursts of gas, but also in their resuscitation by the use of oxygen. It would almost appear from indications in the Mining press, however, as if opinion respecting the future value and application of rescue apparatus were divided into two schools, viz. :—that which expects with perfected apparatus death-rates in mines after explosions to be considerably reduced, and that which is of opinion that such apparatus is as yet on its trial in respect of reliability, and that even assuming its entire reliability, little diminution of death-rates after explosions and underground fires is likely to happen.

On the one hand, it is to be remembered, from the very nature of a colliery explosion, more especially in the future when mines will have to be deeper and underground roads therefore longer, even were a rescue brigade at hand and ready to descend, that some considerable time must elapse before descent is sometimes possible, owing to destruction of shafts and other reasons, during which interval after damp has time to effect its deadly action on the men exposed to it. If, however, descent is practicable, there can be little doubt, assuming a ready brigade to be present, that more lives are likely to be saved than in the past when no such apparatus was available. That salvage of human life, therefore, will depend entirely on the local conditions, and of these possible rapid descent will probably be the chief, since the longer the compulsory delay the greater chance of the mortality from after-damp. With properly-trained and available brigades, therefore, improvement in this respect may reasonably be looked for. This is, perhaps, even more true of underground fires, since without rescue apparatus it would be impossible for men, so unaccoutred, to enter such irrespirable atmospheres. In this direction may improvement be looked for. After explosions, moreover, rescue apparatus are bound to be useful for scouting work, that is, for detecting fires which have resulted from the explosion, for recovering the mine itself by helping to re-establish ventilation, for succouring survivors, and for like purposes.

For fighting fires underground, rescue apparatus has largely been taken advantage of in Germany, and more recently in France and Belgium. Without such apparatus, indeed, it is most difficult to get into touch with the fire, or even to locate its position, and hence it cannot be dealt with effectively. Hitherto underground fires have proved an immense loss to coal-mine owners, since they necessitate large areas of coal being cut off. In districts like Fife in Scotland, and Staffordshire in England, where such fires are not uncommon, rescue apparatus should prove of the greatest value.

Apart from such serious matters, many accidents requiring first-aid assistance occur annually in every mine. Reasonable provisions ought to

be provided, as are laid down in the Statutory Regulations, for such treatment, as well from the point of view of those injured as of the employers. It must obviously be of value to both that injuries are promptly and properly attended to, from the point of view of loss of time off work, and loss of money in compensation for accident.

What is needed for such first-aid treatment at every mine employing a large number of men is a good-sized room, which should contain (1) a large table, (2) two chairs, (3) a mattress, (4) a hot and cold water-supply, and (5) a cupboard for storing dressings and splints. Hot water is essential for bathing injuries, since so many wounds sustained in pits are dirty and oily and require initial careful cleansing. The mattress, for placing on the floor when a patient requires to be placed in the recumbent position for any purpose, such as the administration of oxygen or artificial respiration, or for the application of splints, and for other obvious causes, would be of great service. We believe that such an equipment would quickly repay itself by more rapid cures in many instances.

APPENDIX.

Explosion at West Stanley Colliery, Durham County.— This explosion occurred on 16th February 1909, about 3 p.m., the number of lives lost being 168. The dead bodies of 165 of these were recovered, but two bodies were never recovered. One person, though rescued alive, died subsequently from the ill effects of the gas he had inhaled. The explosion was a double one: that is to say, there were two separate explosions, the earlier one being a mere puff, but the second a very severe one, as judged by the "large ball of fire from the mouth of the pit, followed by a thick black cloud" which issued from the downcast shaft. The verdict of the coroner's jury, based on the evidence given at the inquest, was that the explosion was due to coal-dust.

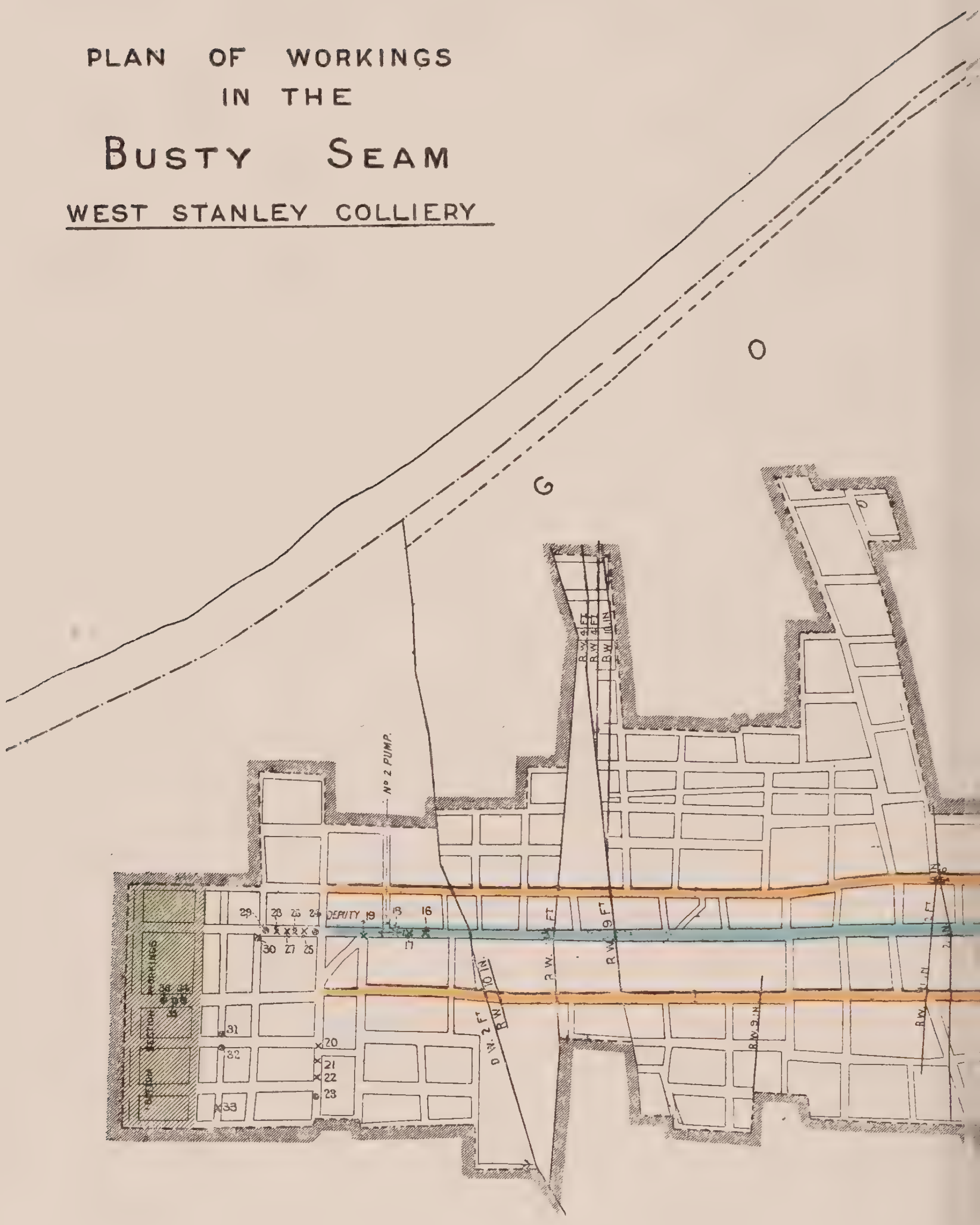
Four seams were being worked in the colliery at the time, viz.— the Towneley, Tilley, Busty, and Brockwell. The coal drawn up the downcast shaft from the Busty level was coal from the Towneley, Tilley, and Busty seams, whilst the coal drawn up the upcast shaft was from the Brockwell seam.

The Brockwell seam was 163 fathoms from the surface, the Busty 139 fathoms, while the others were at lesser depths. The coal is a bituminous coal. The ventilation of the different seams was effected by a Guibal fan, 35 feet in diameter and 10 feet in width, which at 35 revolutions and a water gauge of 1·5 inches was capable of moving from 76,000 to 78,000 cubic feet of air per minute through the mine. The ventilation was proved to be effective shortly before the explosion took place.

The Brockwell seam was worked on the longwall method, and some pillars were being removed, while in the Busty seam the work consisted solely in removing pillars. The workings in all four seams were somewhat dusty, but was perhaps less in the Tilley seam owing to its wetness. The coal-cutting machinery, motors for pumping and other operations, and lighting of the mine, were worked by electricity, the generating plant being, however, on the surface. Watering of the main roads was carried on at frequent intervals, and had been performed in the Brockwell seam on the night before the accident. Shot-firing was carried out in the mine between the hours of 4 p.m. and 4 a.m. In the Busty seam no shot-firing was needed in the coal, and, when needed in the rock, was conducted between the hours named.

PLAN OF WORKINGS IN THE BUSTY SEAM

WEST STANLEY COLLIERY



NOTE.—Thirty-six men were killed in this seam. Evidence of burning a
others. In some of these instances the burning was extremely severe. and s
shattered, and in one the trunk of the body was crushed. In twenty-three
two carbon dioxide poisoning, and in the remainder burning or violent injuries,

found in twenty of these, and of burning coupled with injury by violence in seven
s the force of the violence ; in one case the face was torn, in three the head was
rty-six cases the approximate cause of death was carbon monoxide poisoning, in
mbined.

NOTE.—Forty-eight men lost their lives in this seam. Of these the bodies numbered from two to twenty-eight (exclusive of No. 27) and No. 48, bore more or less evidence of burning. No. 23 and 48 were burned to death, the body numbered one and those numbered twenty-seven, and from twenty-nine to forty-seven, did not show evidence of burning. The cause of death of thirty-seven of the total forty-eight cases was attributed to carbon monoxide, of eight to carbon dioxide, of two to burning, and of one to injuries of the head. On only two bodies of the entire series were there marks of wounding by violence, and in both of these the wounds were on the head.


PLAN OF WORKINGS
IN THE
BROCKWELL SEAM
MAGNETIC MERIDIAN
1906
DEPUTY
R.W.1.0"
R.W.3'.0"
R.N. 2'.0"
D.E. 2'.0"
Two Props had been on fire here
1
2
3
8
9
10
11
12
13
14
15
16
17
18

After the explosion occurred, both downcast and upcast shafts were found to be damaged, although the cages had suffered comparatively little injury. While the work of clearing these shafts was proceeding, the joiner's shop was converted into a temporary hospital, into which medical stores, oxygen, and other things were carried to be in readiness. Draeger and other rescue apparatus was on the ground within two and a half hours of the explosion, and with it were men trained in its use. No opportunity offered itself, however, in which it could have been of any assistance. The temporary hospital had only three occupants, viz.—one man from the Busty seam and two from the Towneley seam. By 2 a.m. of the 17th February, the downcast shaft became available for descent, and rescue parties at once descended into the Towneley and Busty seam workings. From the Tilley seam came 26 men who had been unaffected by the explosion. Their safety was attributed to the wetness of these workings, the comparative absence of dust, and to the fact that these men remained where they were in their places. From the Towneley seam came four men, one of whom, however, after a hard struggle for life for 36 hours, succumbed to the effects of the foul air he had inhaled. One man only was rescued alive from the Busty seam.

The evidence regarding the origin and cause of an explosion being founded upon (*a*) the indications of direction of force, (*b*) the presence of flame, (*c*) the attitudes of the dead bodies and the causes of deaths of the victims, and (*d*) the statements of survivors, the Inspectors of Mines, who inquired as to these and who reported on the disaster, affirm that they have been unable to arrive at any definite conclusion respecting either the point of origin or the cause of this explosion. Suspicion attached to both the Brockwell and the Busty seams: to the former in respect that there was evidence of the presence of fire in some of the pit props in the workings, of coked coal-dust on the floor at two places to a depth of half an inch or more, and of coked dust in the lamps of six victims. Besides, from the attitudes of the bodies of these six men, it was clear that death had overtaken them unwarned. Suspicion was attached to the Busty seam from the evidence of the sole survivor, who stated that he saw the coal-dust being driven in the form of a large cloud after the sound of the explosion, followed by the extinction of the electric lights.

The number of fatalities and the causes of deaths in these several seams were as follows:—In the Towneley seam there were 64 deaths and 3 survivors. Of the 64 dead bodies, 27 showed marks of burning, 3 of these also presenting marks of violence, 3 died from the effects of violence, and 34 showed no evidence of burning. Of the total deaths, the cause in 47 was carbon monoxide poisoning, in 8 carbon dioxide poisoning, and in the remainder, either burning or injuries of a severe

character. In the Tilley seam, there were 18 deaths and 24 survivors. None of these 18 dead bodies showed marks of burning, but 3 of them bore marks of severe injuries from violence, one of these being of a fatal character. The cause of death in 16 of these 18 was carbon monoxide poisoning, in one carbon dioxide poisoning, and in one fatal injuries.

In the Busty seam there were 36 deaths and one survivor. Evidence of burning alone was found in 20 of these, and of burning coupled with injury by violence in 7 others. In some of these the burning was extremely severe, and so also in others was the force of the violence. In one case the face was torn, in 3 the head was shattered, and in one the trunk of the body was crushed. In 23 of the 36 cases the proximate cause of death was carbon monoxide poisoning, in two carbon dioxide poisoning, and in the remainder, burning or violent injuries or both combined.

In the Brockwell seam 48 men lost their lives. Of these, 27 showed marks of burning, 2 of them being burnt to death, while in two others of this series injuries as well as burning were found. No evidence of burning was found on 21 of the entire number. The cause of death of 37 of the total number of bodies was attributed to carbon monoxide poisoning, of 8 to carbon dioxide poisoning, and of two to severe burning. On two only of the bodies were there marks of wounding by violence, and in both of these the wounds were on the head.[1]

MINE RESCUE REGULATIONS.

After the foregoing pages were printed, a Circular Letter was issued from the Home Office of date 16th April 1914, stating that in consequence of objections taken by the Mining Association of Great Britain and the North of England Coal Trade Association to the provisions as to rescue appliances contained in Part IV. of the General Regulations of 10th July 1913, the question was referred to a Referee, and at the hearing before the Referee an arrangement was suggested which appeared to the Secretary of State to be a good one, and which has now the assent of the representatives of both owners and miners. Under this arrangement the use of self-contained breathing apparatus will be made compulsory; but an alternative system for the establishment of rescue brigades will be admitted by which, in place of local brigades at the collieries now required, permanent brigades will be maintained at the Central Station,

[1] Report on Explosion in West Stanley Colliery. Redmayne and Bain, H. M. Inspectors of Mines, 1909. (*See* Plans opposite p. 436.)

and one or more men, according to size of colliery, will be trained at each mine to act with the Central brigade when summoned to the mine.

The Home Secretary has accordingly issued fresh draft amending Regulations of date May 1914, which, if accepted, the draft Regulations of Nov. 1913 will be withdrawn.

The following is the substance of the new draft Regulations :—

1. Nos. 140, 141, and 142 of the Regulations above-mentioned (*Cf. pp.* 421-425 *of text*) shall not apply to any mine served by a central rescue station with a permanent rescue corps, and which is situated within a radius of 10 miles, and is in telephonic communication with it, subject to the following conditions :—(*a*) The central station rescue corps shall consist of not fewer than six men, or if the total number of underground employees at all mines served by the station exceeds 15,000, eight men; the members of such corps to be selected men, medically certified to be fit for such work, and each to be the holder of a certificate of proficiency in first-aid from a body recognised by the Home Secretary. The members of such corps shall be continuously employed and in constant residence at the station. The corps shall be thoroughly trained in the use of breathing apparatus and in rescue work, and shall be constantly kept in a highly efficient state. One or more members of such corps shall be appointed to act as leaders. (*b*) One or more persons employed at each mine shall be selected for the purpose of acting with the rescue corps from the central station when summoned, as follows :— if the total number of underground employees is not less than 100 and not more than 250, *one* person; if more than 250, and not more than 1000, not less than *three* person; and if more than 1000, not less than *five* persons. These persons shall be thoroughly trained and shall from time to time undergo practices at the central station in the use of breathing apparatus and in rescue work, shall be carefully selected as in the case of the rescue corps at the central station, and so far as reasonably practicable at least one person shall be selected from each shift. Arrangements shall be made for summoning such persons immediately their services are required. No breach of condition (b) shall be deemed to have arisen in consequence of failure to maintain the full number of trained men in accordance with the foregoing provisions, if the owner, agent, or manager of the mine satisfies the Inspector of the Division that he has made every effort to comply with those provisions, and that the failure was due to causes over which he had no control. (*c*) Tracings of the workings of the mine are to be kept as stated on p. 422. (*d*) There shall be provided and maintained at the mine :—(i) two complete suits of breathing apparatus or two smoke helmets (that is, appliances for supplying fresh air to the user by means of a pipe and bellows), or one of each in an efficient state and constantly ready for immediate

use, (ii) two or more small birds or mice for testing for carbon monoxide, (iii) one electric hand-lamp and one safety-lamp for testing for fire-damp for each person trained in pursuance of condition (b), and (iv) one oxygen reviving apparatus.

Definition of *breathing apparatus.*—For the purpose of the foregoing regulation and of Part IV. of the General Regulations above-mentioned, breathing apparatus means an apparatus of such a character that the wearer carries with him the means for respiration in an irrespirable atmosphere, and is not dependent for them, while in such an atmosphere, on any other person or persons.

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